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Book Reviews.

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CALCIFICATION OF THE CEREBRAL VESSELS WITH A CLINICAL PICTURE SIMULATING BRAIN TUMOR*

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INTRODUCTION

In this paper we offer a somewhat detailed clinical and histologic report of a case which is interesting in three respects: First, a diagnosis of brain tumor was clinically justifiable and several operations were performed in the hope of removing the supposed tumor. In other words, it was an example of so-called pseudotumor cerebri. Second, epileptiform attacks of petit mal and grand mal types were the principal symptoms, so the case shows what kind of changes occasionally may cause such attacks. Third, and chiefly, the case is of interest on account of the most unusual pathologic condition presented; namely, a large indurated area in the interior of the left hemisphere in which the capillaries were infiltrated with a hyaline, or colloid material and also calcified to such an extent that decalcification was necessary for the preparation of suitable microscopic specimens.

While calcification of large sclerotic vessels is extremely common as a general process, and while calcification of a single vessel, such as the renal, coronary, or femoral artery is not rare, there are few reports of a condition at all similar to that presented in our case. Mallory [1] relates the case of a woman, aged 45 years, with a clinical diagnosis of chronic nephritis, in which there was colloid and calcareous infiltration of the cerebral vessels, the lime salts being found only in the colloid

*From the pathology laboratories of the Illinois State Psychopathic Institute and Cook County Hospital.

* Read before the Section on Nervous and Mental Diseases at the Seventy-Second Annual Session of the American Medical Association, Boston, June, 1921.

1. Mallory, F. B.: A Contribution to the Study of Calcareous Concretions in the Brain, J. Path. Bacteriol. 3:110, 1894.

material. Hansemann [2] reports the case of a man, 28 years old, who had suffered from a speech disturbance, and toward the end also from convulsions, dizziness, headache and vomiting. Colloid degeneration and calcification were present in the vessels of the white matter of both hemispheres, while the large cerebral vessels and all vessels outside the brain were normal. Arnold Pick's [3] patient was still younger, 24 years old, and had suffered from epilepsy from childhood. Later,

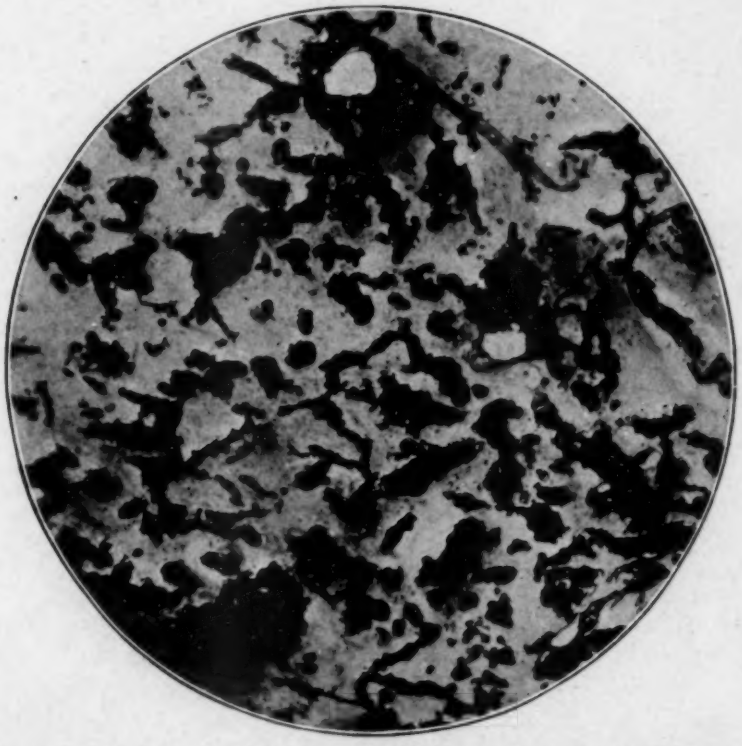

Fig. 1.—A calcified area from the left centrum semiovale in the region of the parietal lobe. Some calcified formations show distinctly a network of petrified capillaries, some are a shapeless mass. Picture under higher power in Figure 2. Thionin stain; X 85.

stupor and signs of tetany were present and brain tumor was suspected. The large vessels were normal, the smaller ones calcified. Similar isolated involvement of larger cerebral vessels is discussed by Marburg,[4]

2. Hansemann: Ein casuistischer Beitrag zur Verkalkung der Gehirngefässe, Verhand. der deutschen pathologischen Gesellschaft **2**:399, 1900.

3. Pick, A.: Calcification of the Finer Cerebral Vessels, with Remarks upon Its Clinical Significance, Am. J. Insan. **61**:417, 1905.

4. Marburg, Otto.: Zur Pathologie der grossen Hirngefässe, Wiener klin. Wchnschr. **15**:1216, 1902.

while Schmincke[4a] reports calcification of smaller and middle sized vessels of the cortical and subcortical regions in two cases (patients 9 and 18 years old).

Much has been written about "pseudotumor cerebri." One of the best studied cases has recently been reported by Percival Bailey.[5] The symptoms led to exploration for suspected cerebellar tumor, but necropsy proved the case to be one of hemorrhagic meningo-encephalitis

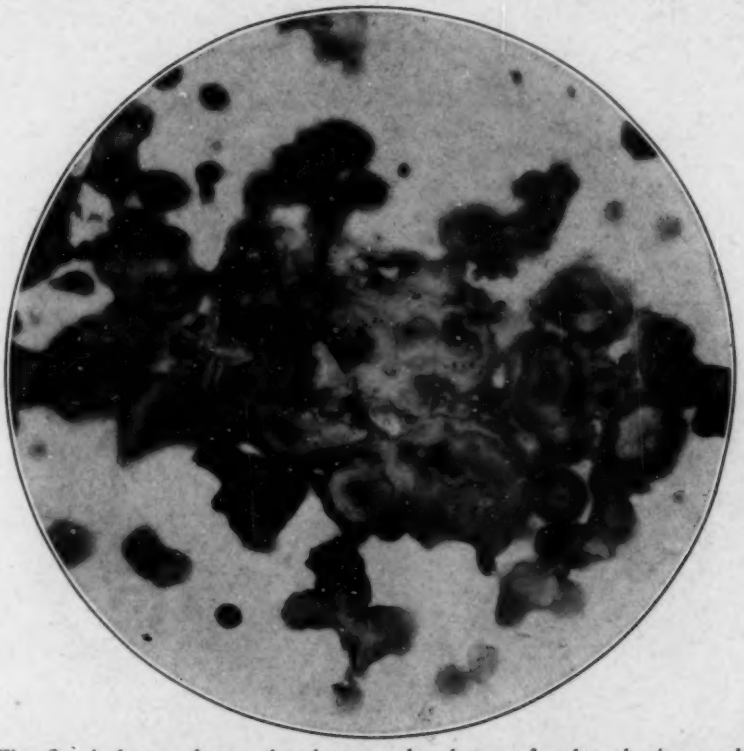

Fig. 2.—A large plaque showing an abundance of calcospherites and capillaries surrounded by a homogenous colloidal substance. One of the areas shown in Figure 1 decalcified with glacial acetic. Van Gieson stain; X 210.

with greater involvement of the cerebrum than of the cerebellum. Bailey also gives a concise review of the "pseudotumor" problem and of the more important cases in the literature.

CLINICAL REPORT OF A CASE (DR. BASSOE)

History.—A business man, married, 32 years old, was referred to me by Dr. J. A. Denne, on April 25, 1911. The family history was negative.

As a child he had a "weak back" but later became strong. He had typhoid at the age of 21. He had had frequent epistaxis for years until the beginning of the

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4a. Schmincke, A.: Encephalitis interstitialis Virchow mit Gliose and Verkalkung, Ztschr. f. die ges. Neurol. u. Psychiat. **60**:290, 1920.

5. Bailey, Percival: Contribution to the Histopathology of "Pseudotumor Cerebri," Arch. Neurol. & Psychiat. **4**:401 (Oct.) 1920.

convulsive seizures. He had had no trauma or venereal disease. He used neither alcohol nor tobacco.

The patient began to have light convulsive attacks in March, 1909, which became very frequent, usually from six to eight daily. The first attack was said to have occurred in the night, when he woke up "with a sensation of blood coming out of all the pores on the right side of the face and of the circulation being shut off in the right arm and leg." He could not talk plainly for about half an hour. Such attacks occurred every day. During these he had a vacant, staring expression, and did not answer if spoken to. If he walked during the attack, there was some scuffing of the right foot. The first attack with loss of consciousness occurred in May, 1910, one in February, 1911,

Fig. 3.—A larger vessel of the subcortical area. The Virchow-Robin space is packed with lime salts; the intima and adventitia are well developed, the latter being hyperplastic; the vessel is surrounded by an empty free space, known as the space of His. The latter is bordered by a thickened layer of glia tissue (upper and right parts of the illustration). The rest of the illustration shows numerous glia cells. Bielschowsky's stain; X 170.

and one in April, 1911. Biting of the tongue and frothing at the mouth occurred during these attacks, and once he fell out of bed. The convulsions began in the right side and then became general. Headache and vomiting were not complained of.

Examination (April, 1911).—The pupils and eyegrounds were normal; there was no gross change in the visual fields. The right knee jerk was slightly stronger than the left; the other tendon reflexes were equal and normal. The right abdominal reflex was slightly weaker than the left. The plantar reflexes

were normal. Sensation, including stereognostic sense, was normal. The Wassermann test of the blood was negative.

Subsequent History and Treatment.—When seen again in November, 1912, he stated that objects sometimes would fall out of his hand in light attacks, and the right ear felt as if plugged. Involuntary urination sometimes occurred. The right abdominal reflex was now absent, and the wrist and elbow jerks were stronger on the right side. Dr. Brown Pusey found that the poor vision in the left eye, of which he had always complained, was due to hypermetropic astigmatism. The patient complained of trembling of the right hand and difficulty in writing and shaving. In December, 1912, the right abdominal reflex was again obtained, but was weaker than the left one. The cremasteric reflex was present

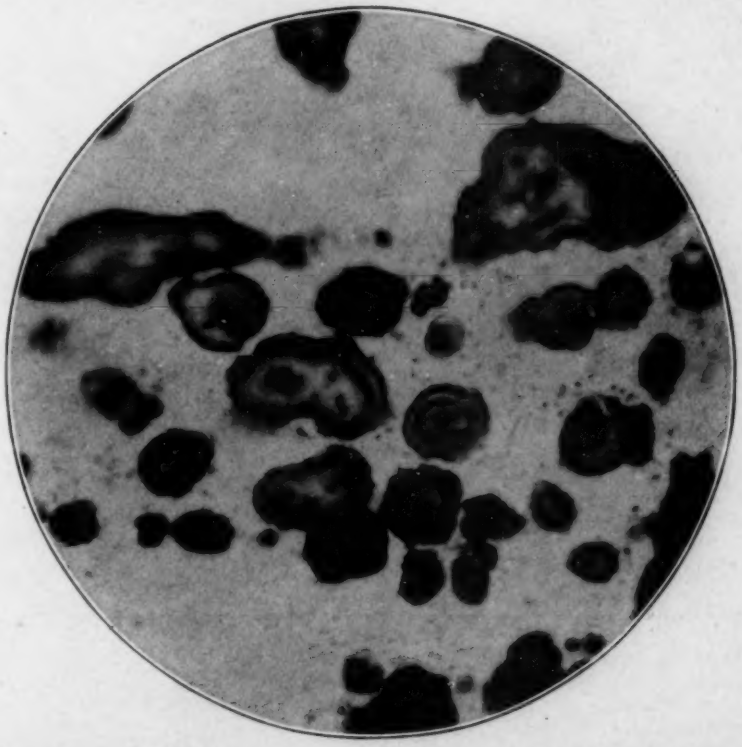

Fig. 4.—A group of psammoma bodies or calcospherites. In some the lamellated structure is marked, some are fused forming larger plaques invested with distinct homogeneous membranes. Some plaques show an abundance of small formations which have no definite structure, such as that of bone or other cell bodies. They evidently are remnants of former capillaries which are quite distinct in the center of isolated calcospherites. The fusion of the latter and formation of plaques can be seen in some areas. Hematoxylin-eosin stain; X 220.

on both sides. The spinal fluid was clear, under normal pressure, gave a negative Wassermann reaction and there was no increase in cells. Six major attacks were observed during the first seven months of 1913. In the lighter attacks he would walk about, dragging the right leg, and would jerk the right arm and shoulder and throw the head to the right.

The first operation was performed by Dr. Dean Lewis on Aug. 29, 1913. An osteoplastic flap over the left motor area was turned down. The dura was tense, but the surface of the brain appeared normal. On introducing a needle, resistance was felt one and one-half inches from the surface. After the operation, the right side was weak, and he was aphasic for a few weeks. Later, his condition was as before the operation except that he could use his right hand better, particularly in writing. In October, 1913, his wife reported that he had had several severe attacks during sleep, and that each time he had been lying on his left side. During this time marked pulsation and a bruit appeared at the lower end of the craniectomy wound. In 1914, he had had comparatively few severe attacks and was regularly taking sodium bromid

Fig. 5.—Several decalcified (with glacial acetic acid) capillaries surrounded by a homogeneous area of a colloid substance. The larger capillary is branched and shows an abundance of lime droplets over the walls. Toluidin blue; X 425.

with a little Fowler's solution. The latter was probably responsible for the marked pigmentation of the skin and keratosis of the palms which appeared during the year. In February, 1915, he was sent to Dr. Harvey Cushing.[6] The most noteworthy features at that time were the right-sided weakness, distinct hesitation in speech and general pigmentation. Slight hyperemia and a little blurring at the upper pole of the right disk were noted, and the left was

6. We are greatly indebted to Dr. Cushing for placing his complete record of the case at our disposal.

edematous. The visual fields were normal. All forms of cutaneous sensation were slightly diminished on the right half of the entire body. The patient gave evidence of impairment of memory and of being somewhat euphoric and silly. Spontaneous speech was slow; he frequently hesitated for words and occasionally used the wrong word. The abdominal and plantar reflexes were not elicited, while the knee jerks were active and equal. The right arm was weaker than the left. The grip of the right hand measured 25 kg. and the left, 31 kg. In addition to the slight right hemianesthesia mentioned, there was also diminished stereognostic sense in the right hand. A soft, blowing systolic murmur was heard at the base to the left of the sternum. The blood pressure was: systolic, 178; diastolic, 118.

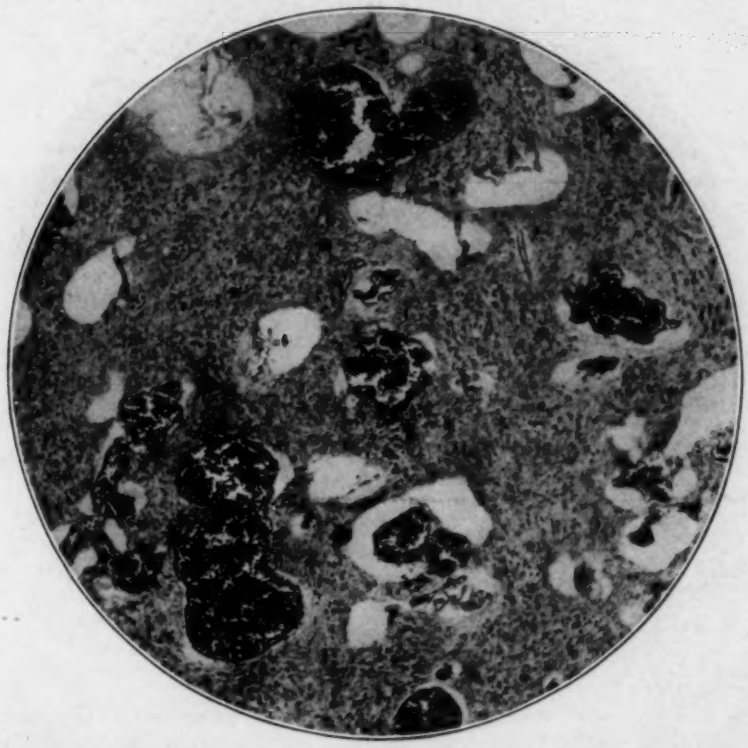

Fig. 6.—An area bordering on the calcified portion of the white substance. A number of hyperemic vessels, mostly veins, fill the large vacuoles, some of which are empty and some of which contain bloodless capillaries. Bielschowsky stain; X 85.

On Feb. 13, 1915, Dr. Cushing performed an operation which consisted of resection of an arteriovenus aneurysm of the temporal vessels, which had developed after the previous operation, and removal of an area of bone posterior to the old wound. The operation was continued on February 19. It was exceedingly difficult to reelevate the old flap and, on account of great loss of blood, the operation had to be abandoned before any subcortical exploration could be made. The patient returned to his home in March, 1915. The attacks

continued, and mental deterioration progressed so that he had to give up his work. In January, 1916, he returned to Dr. Cushing. The right-sided weakness and anesthesia were more marked than before.

Ophthalmoscopic examination by Dr. Walker showed that the right disk was paler than the left one and that the margins were slightly faded. The lamina cribrosa was obscured, apparently by a layer of new tissue. There was slight perivascular streaking. The veins had slight residual tortuosity. The conditions suggested a previous inflammation. There was possibly some elevation of the disk.

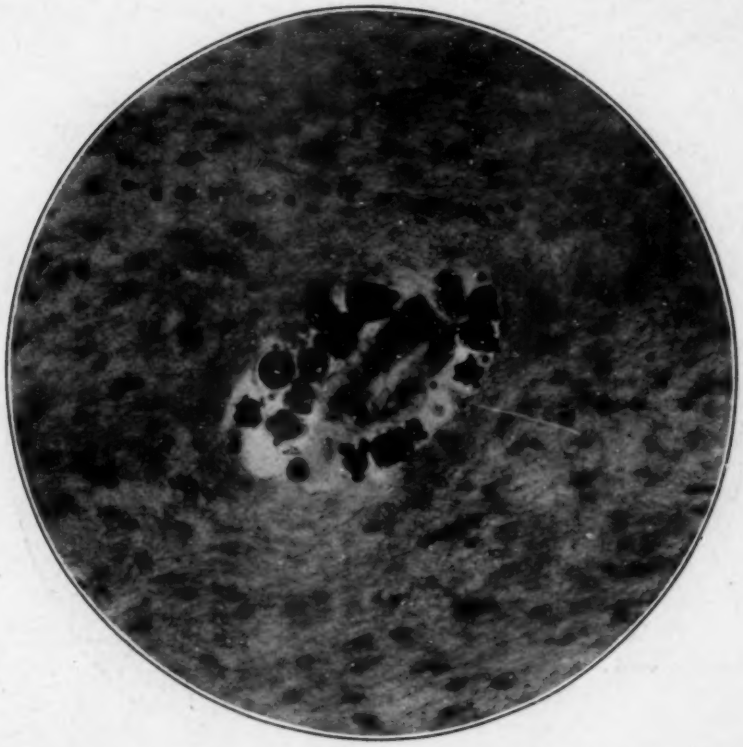

Fig. 7.—A type of small blood vessels scattered over the brain substance. The adventitia is thickened and proliferated, containing in its distended so-called lymph space an abundance of gitter cells packed with large and small drops of fat. Marchi counterstained with Alzheimer-Mann; X 275.

Another operation was performed by Dr. Cushing on Feb. 4, 1916. This time a flap was successfully turned down. The convolutions were flattened and, on palpation, somewhat soft. A needle was inserted in three separate situations and posteriorly it came down to what seemed to be a resistant mass, possibly a tumor. Anteriorly the needle passed without resistance. Although these three punctures were carried down to the depth of the ventricle, no fluid was secured, which led to the inference that the ventricle was compressed and that a tumor probably was present somewhere in the hemisphere. An incision was made posteriorly and carried down to the depth at which resistant tissue had been encountered with the needle. No definite tumor was seen, but

the brain tissue was denser and firmer than normal. A small piece of this dense white matter was removed from a depth of about 4 cm. The wound was closed, and the patient made a good operative recovery. Examination of the small piece of tissue removed merely revealed marked gliosis but no evidence of tumor.

There was some subsequent increase in the right-sided weakness, but the attacks for a time were somewhat milder. The mental deterioration increased.

The patient died at his home in May, 1920. Necropsy examination was limited to the brain which was sent to me.

Fig. 8.—Gray substance of corpus striatum; a branched blood vessel with proliferated adventitia surrounded by a dense wall of glia nuclei. The substance shows an abundance of ganglion cells surrounded and invaded by glia cells. Toluidin-blue stain; X 190. (Use hand lens.)

PATHOLOGIC REPORT (DR. HASSIN)

The brain was received in formaldehyde solution. It had been incised when fresh and placed in a small container so that it was in poor condition for macroscopic study. It weighed 1,440 gm. There was a discolored, yellowish area in the pia of the left temporal lobe, and the underlying cortex showed punctate hemorrhages. The convolutions as a rule appeared rather flattened and the sulci shallow. The arteries at the base showed little, if any, thickening. The cerebral ventricles were small. No gross changes were discernible in the right cerebral hemisphere, cerebellum, pons or medulla. On making additional

incisions into the left cerebral hemisphere a leathery resistance was encountered and in many places a gritty sound produced. The indurated area measured about 2 by 3 cm. and mainly occupied the basal ganglions and centrum semiovale. It was ill-defined, merging into the surrounding normal substance. The cortical gray matter appeared normal as did the ependyma and choroid plexus.

Microscopic Studies

The microscopic studies covered every portion of the brain. Celloidin, paraffin and frozen sections were stained by numerous methods, such as thionin, hematoxylin-eosin, methyl-blue eosin, Marchi, Bielschowsky and others.

Fig. 9.—White substance (centrum semiovale) showing a black area of fat; the other half shows a few fat droplets (dark spots) and pale calcified bodies invested with a distinct membrane. Herxheimer's scarlet red-hematoxylin stain; X 85.

The white substance, containing the indurated islands, showed numerous homogeneous, shapeless formations which, in some places, occupied the entire field (Fig. 1), totally obliterating the parenchyma. Some sections exhibited more distinct formations in the shape of single round or oval bodies, or of large islands, plaques or conglomerates (Fig. 2). In some places, a distinct network of capillaries could be made out, while in others there were abundant drops and droplets scattered over the parenchyma and often gathered near the blood vessels, constricting their lumen or covering their walls and giving them a marked dotted appearance. Some capillaries were entirely made up of such

rows of droplets, while others were only partially covered by them, the droplets evidently becoming confluent and transforming a portion of the capillary into a structureless mass.

The larger vessels (arteries and veins) usually exhibited a normal structure. Some showed within the adventitia the same amorphous substance as that contained in the capillaries. It was confined to the adventitial spaces of Virchow-Robin (Fig. 3), the adventitia being markedly hyperplastic and often showing as a thick homogeneous fibrous ring.

These four types of the amorphous substance—large solid masses forming islands, isolated drops and droplets in the parenchyma, droplets over the capillaries, infiltrations of the adventitial spaces—though quite recognizable in

Fig. 10.—Right Ammon's horn. Satellitosis and neuronophagia. Many normal hyperemic capillaries, many are newly formed; absence of perivascular infiltrations. Thionin stain; X 370.

the unstained specimens, were especially distinct when stained. Of the numerous methods used, only that of von Kossa[7] (2 to 5 per cent. of silver nitrate) left them unstained, while Bielschowsky's method (ammonia silver) stained them beautifully. Acids, such as prolonged action of 5 per cent. nitric, hydrochloric or glacial acetic dissolved them with some difficulty, without the formation of bubbles, or rendered them very transparent. Doubtless, we had here to deal with lime deposits. Stained decalcified specimens revealed a great number of

7. Von Kossa, J.: Ueber die im Organismus künstlich erzeugbaren Verkalkungen, Ziegler's Beitr. z. Path. Anat. **29**:163, 1901.

round or slightly oval bodies in the form of concentric rings which very much resembled so-called psammoma bodies, corpora arenacea or calcospherites, also known as Harting's bodies (Fig. 4). These bodies were of various sizes, always invested by a thin homogeneous membrane, were scattered singly or formed more or less voluminous islands of irregular shape. The membrane closely followed the contour of the islands and separated them from the surrounding parenchyma. These calcospherites did not give the amyloid reaction, that is to say, they did not stain either with iodin or iodin sulphuric acid, neither did they take the fat stains (scarlet red, osmic acid), but were very well stained with every basic stain and by Bielschowsky's method.

Fig. 11.—Pia-arachnoid with hyperemic veins and meshes infiltrated with blood cells and other elements described in the text. Alzheimer-Mann stain; X 27.

In the capillaries the decalcified specimens showed equally noteworthy features. Photomicrograph 5 shows capillaries possessing a distinct lumen, their walls dotted and surrounded by a broad pale homogeneous substance very much resembling colloid or hyalin. Of the same character is the substance of the islands or conglomerates of the calcospherites in which it occupies a much larger area than the network of the capillaries. The latter are distinctly seen to be the nuclei or centers of the calcified masses.

Aside from vessels surrounded by the amorphous colloidal substance, there was a great number of blood vessels free from any foreign material, but densely hyperemic (Fig. 6) and possessing a thickened wall with well stained

nuclei. Especially numerous were such vessels in the areas adjacent to the calcified blood vessels, while other areas even remotely situated from those of calcification showed capillaries infiltrated with gitter cells packed with fat (Fig. 7). Often they were surrounded, muff-like, by a dense ring of delicate glia fibers, or by an enormous mass of glia nuclei, frequently in the form of long rows resembling a wall around the vessel (Fig. 8). Many vessels had thickened adventitia and a greatly narrowed or almost obliterated lumen, while in other places, particularly in the calcified areas, no substance whatever was found, the field appearing vacuolated or areolar.

Areas bordering on those containing the calcified masses, when stained with scarlet red, showed large amounts of lipoid, or fatlike substances, covering the entire field, including the perivascular spaces and the glia, but sparing the nerve fibers (Fig. 9). Such areas appeared altogether red, in sharp contrast to the neighboring blue calcified masses.

Some areas of the white as well as of the gray substance of the large basal ganglions contained numerous foci of fresh and old hemorrhages with a great mass of proliferated adventitial cells, rod cells ("Stäbchenzellen"), plasma cells and pigment granules, mostly contained within macrophages and surrounded by dense walls of glia nuclei. Other areas presented miliary foci of degeneration consisting of sclerosed vessels surrounded by a great mass of gitter cells and macrophages.

Most of the ganglion cells of the basal ganglions in the neighborhood of the calcified areas were normal. However, some showed an abundance of lipoids and neuronophages, the surrounding glia cells being unusually rich in chromatin and often forming clusters (Fig. 8). Among the masses of glia cells were scattered nerve fibers which were thickened, spindle shaped, tumefied, twisted or broken up. The ependyma of the ventricles was proliferated, the subependymal layer consisting of rarefied glia and great masses of glia cells.

Summary of Changes in the White Substance and Basal Ganglions.—The changes found were: calcium deposits around the capillaries of the white substance, calcareous infiltration of the adventitial spaces of the larger vessels, reactive proliferative phenomena in the glia, ependyma and larger vessels, foci of hemorrhages and degeneration in both the gray and white substances and mild parenchymatous changes in the ganglion cells as well as in the nerve fibers.

Cortex.—Comparatively slighter were the changes in the cortex though satellitosis and neuronophagia were quite pronounced. Photomicrograph 10 shows an area from the cornu ammonis densely covered with clusters or islands consisting of ganglion cells invaded or surrounded by glia nuclei.

The white subcortical matter contained an abundance of ganglion cells poorly stained, greatly reduced in size and with shrunken processes. Marchi and scarlet red stains showed fat and pigment in every ganglion cell, as well as in many perivascular spaces. The vessels showed no calcareous deposits, but were greatly hyperemic and often surrounded by walls of glia nuclei, and were decidedly proliferated. In the parietal lobe there was a great number of old capillary hemorrhages such as those noted in the basal ganglions, while in the deeper strata there were congregates of vessels with an abundance of greenish pigment in their adventitia. The minimum changes were present in the frontal lobes, the most pronounced in the cornu ammonis and in the parietal lobe, while the paracentral lobe showed hardly any change. Neither did the cerebellum, pons or corpora quadrigemina exhibit any changes, except some vascular proliferation.

Summary of Cortical Changes.—There were slight parenchymatous changes combined with mesodermic ones, such as hyperemia, vascular proliferation and miliary hemorrhages. As compared with the changes in the basal ganglions, the cortical ones were insignificant.

Pia-Arachnoid, Choroid Plexus and Large Cerebral Vessels (Especially of the Base).—The pia all over the cerebral cortex showed (Fig. 11) distended meshes containing scattered blood cells, numerous foci of hemorrhages and an abundance of macrophages packed with blood pigment and red cells. There were also many mononuclear leukocytes (polyblasts) rich in cytoplasm with more or less intensely stained nuclei and typical plasma cells. The hemorrhagic foci were particularly numerous in the region over the parieto-temporal lobe, less so over the frontal lobe, in which only a few macrophages could be seen loaded with pigment. The vessels of the pia were distended, hyperemic and surrounded with blood pigment but never infiltrated, although the walls appeared thickened, containing oblong nuclei, were well developed and packed with pigment granules. Pigment and some red cells could also be found in the arachnoid membrane which was rich in connective tissue fibers, fibroblasts and clusters of mesothelial cells. Other cells were distinctly reticular in structure much resembling gitter cells. This membrane appeared much thickened, brightly stained, and, like the pia, exhibited marked proliferative phenomena and evidences of old hemorrhages. In addition, both the pia and arachnoid showed abundance of fat granules (Marchi stain).

The choroid plexus showed dense infiltration of its stroma, engorged blood vessels, many of which were calcified, with the lumen entirely obliterated. The epithelial cells covering the blood vessels were of large size and vacuolated, contained an excentric nucleus, black granules and some amorphous substance. A careful study of the basilar and vertebral arteries showed no changes.

GENERAL SUMMARY AND DISCUSSION

The changes outlined were pronounced in the mesodermic tissues (pia-arachnoid, capillaries and larger vessels) and somewhat less marked in the parenchyma (ganglion cell changes and foci of degeneration). Both parenchymatous and mesodermal changes were associated with old foci of hemorrhages and vast deposits of lime (calcifications). The hemorrhages were accidental, the result of three bloody operations. The calcifications were confined to the capillaries of the centrum semiovale and basal ganglions, while the larger vessels only occasionally showed the presence of calcareous masses which were limited to the adventitial spaces. The condition of both the capillaries and larger vessels was altogether unique and different from that observed in syphilis, endarteritis obliterans, so-called calcium metastases, arteriosclerosis and purely inflammatory states. As noted, the calcareous deposits occupied areas infiltrated with a colloidal or hyaline substance; sometimes they occurred in the form of droplets on the surface of the capillary walls, or totally enveloped and destroyed them. In far advanced stages, they formed calcospherites containing broken-up strands and fibers of connective tissue which probably were the sole remnants of former capillaries. As the adventitial spaces of normal,

as well as of calcified, capillaries are in direct communication with those of the larger vessels, it is to be expected that a certain amount of the calcareous substances would be transported to the latter and deposited in their adventitial spaces. The presence of lime salts in the latter would provoke hyperplasia of the adventitia, as well as proliferation of its nuclei, and ultimately lead to fibrous thickening of the vessel wall.

The presence of lime salts in the walls of larger vessels of the brain substance is thus a secondary process which is also true of the capillaries. Being in intimate anatomic association with the tissues, the capillaries are the first to suffer if pathologic phenomena take place in the tissue spaces, tissue fluids or the tissues themselves. If these formations happen to undergo chemical or other changes, such as deposits of colloidal substance, the latter will accumulate around the capillaries, will not be drained by their adventitial spaces, will become stagnant, anchored as it were within the tissues and like any other useless or dead material will ultimately become infiltrated with lime salts. We, therefore, must assume that the tissues harboring the capillaries, as well as the tissue spaces, are the primary sites of the calcareous deposits, and that the calcification is preceded by a stage of some degeneration (colloidal or hyaline). In some cases calcification does not follow the degeneration stage, and the entire pathologic condition is then confined to the stage of colloidal degeneration. This has been demonstrated by Alzheimer,[8] who considers the tissue fluids to be the principal places in which the colloidal masses originate.

These masses are, therefore, to be looked on as a preliminary stage of calcification, an opinion long ago expressed by Holschewnikoff[9] and Mallory, though the former thought that they originate, not in the tissue fluids, but in the capillary endothelium which subsequently undergoes calcification. On the other hand, von Kossa's experiments with calcification of renal arteries in animals led him to believe that the tissue fluids are the places in which lime salts are deposited. The foregoing considerations led us to the conclusion that calcareous deposits, such as were found in our case, are not indicative of some primary vascular disorder, as was thought by Pick and others, but are evidence of some metabolic disorder of the tissue fluids themselves.

The damaged capillaries invaded and covered by calcareous masses were, of course, incapable of nourishing the surrounding tissues. This resulted in necrobiosis with the formation of numerous foci of secondary

8. Alzheimer, A.: Die Colloidentartung des Gehirns, Arch. f. Psychiat. **30**:118, 1898.

9. Holschewnikoff, D.: Ueber hyaline Degeneration der Hirngefässe, Virchows Arch. f. path. Anat. **112**:552, 1888.

nerve degeneration and the accumulation of large masses of lipoid substances. These lipoid masses drained by the adventitial spaces were carried to the cerebral subarachnoid space, in which they could easily be demonstrated with appropriate stains (osmic acid, scarlet red).

The two foregoing pathologic conditions—calcification and fat accumulation—were associated with purely inflammatory phenomena, such as vascular hyperemia, new formation of capillaries, ganglion cell and glia changes, while infiltrative phenomena were in the background. Such a type of brain inflammation, known as productive encephalitis, is characteristic of toxic states caused by organic or inorganic poisons, such as lead and arsenic. It is highly probable that both the encephalitis and the colloidal degeneration, with the subsequent calcification, were due to the same cause—some severe intoxication.

The latter also plays a prominent rôle in causing so-called ameboid degeneration of the glia, as well as of some gliomatous growths. The ameboid glia strikingly resembles in its physical-chemical aspects colloidal masses, the similarity being so striking that it is impossible to tell the difference under the microscope. Whether or not these two substances are identical is hard to prove, but it is rather suggestive that both are a manifestation of some deep catabolic disorder. Such a disorder probably is also the principal cause of calcification in other regions, the arachnoid, for instance, in which the calcium deposits occur not around the capillaries, but, as demonstrated by Cushing and Weed,[10] in the cells themselves, principally in their nuclei. The conditions in the arachnoid, of course, are different form those in richly vascularized brain tissue; yet the calcification, even in the arachnoid, must be a secondary process, following degeneration of the arachnoid cells.

Wells[11] ably took up the purely chemical problems in connection with calcification in general. He came to the conclusion that calcification is rather a "physico-chemical process beginning as a simple adsorption by hyaline substances which have a more or less specific absorption affinity for calcium." It is, of course, impossible to tell why the abnormal and as yet unknown processes ultimately leading to calcification should affect the white substance only, especially that of the large ganglions, just as it is impossible to explain why these regions are the favorable sites for other abnormal processes, such as Wilson's disease and similar syndromes.

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10. Cushing, Harvey, and Weed, Lewis H.: Studies on the Cerebro-Spinal Fluid and its Pathway, No. IX. Calcareous and Osseous Deposits in the Arachnoidea, Johns Hopkins Hosp. Bull. **24**:366, 1915.

11. Wells, H. Gideon: Calcification and Ossification; Harvey lecture, Delivered March 25, 1911, Arch. Int. Med. **7**:721, 1911.

CONCLUSIONS

1. The white substance of the brain, especially that of the basai ganglions, may be the site of a peculiar degenerative process, followed by calcification.

2. This process is confined to the capillary system.

3. The larger vessels show the same deposits within their adventitial spaces which are invaded secondarily from similar spaces of the calcified capillaries.

4. The lime deposits originate in the tissue spaces which primarily show a hyaline or colloidal degeneration, as the result of some metabolic disorder.

5. The degeneration results in defectvie drainage of the tissue fluids which become stagnant and ultimately calcified.

6. The process is associated with widespread and varied changes in the cerebrum and most likely in the pia-arachnoid. The precise condition of the latter could not be ascertained in our case in view of the traumatic hemorrhages.

7. Clinically, a peculiar mental and nervous condition may obtain which suggests brain tumor.

ABSTRACT OF DISCUSSION

Dr. Percival Bailey, Boston: I recently had an opportunity to examine in Dr. Hassin's laboratory, a brain from a patient operated on by Dr. Harvey Cushing. The patient had had a complete syndrome of the left cerebellar hemisphere. He died some time later, and the brain was sent to me for examination. There was found a meningoencephalitis. In every case examined since Nonne's original communication on "pseudo-tumor cerebri," definite pathologic changes have been found. Dr. Bassoe and Dr. Hassin now establish the pathologic changes in another case. The term "pseudo-tumor" should be abandoned, since it has no clinical or pathologic significance.

Dr. Walter Timme, New York: I was particularly interested in the cases Nonne described in his account of pseudo-tumor cerebri. The pathogenetic factor in some of his cases was a lead encephalopathy as shown by Maas in cases of necropsy. One man was a typesetter; a woman was the wife of a man engaged in the lead industry. There was typical choked disk and all the symptoms of tumor cerebri. Dr. Bassoe, will you give us your findings in cases of lead poisoning?

Dr. Peter Bassoe, Chicago: I believe many of these cases of so-called pseudo-tumor really represent vascular lesions. I recall another patient sent by me to Dr. Cushing for operation in whom only an area of thrombotic softening was found although there was no reason to suspect vascular disease. Another case was that of a man I saw eleven years ago, who presented the symptoms of general increase in cranial pressure including choked disks but no focal symptoms. There was apparent recovery after a decompression and from that time on there was no suggestion of brain lesion, but the patient later developed

symptoms of angioneurotic edema. The question arises: May this condition cause symptoms of brain tumor? In regard to Dr. Timme's question about lead encephalopathy, Dr. Hassin made a report at the meeting of the Research Association in Nervous Diseases and spoke on the pathology, which is characterized by proliferation of the small vessels. If we assume that this process is localized, it is easy to understand how a syndrome similar to that of a tumor may be produced.

THE INTRANEURAL PLEXUS OF FASCICULI AND FIBERS IN THE SCIATIC NERVE*

JOHN CHARNLEY McKINLEY

MINNEAPOLIS

During the past fifteen years various writers have developed the conception of fiber tracts within peripheral nerves analogous to those in the spinal cord and brain. That is, in a given nerve, the fibers that supply a certain muscle or a certain area of the skin have been thought to be situated constantly within a definite region of the trunk of the nerve at a given level. Thus, the anatomist Bardeen,[1] from his observations on the segmental distribution of the peripheral nerves of the lower extremity in the embryo and his dissections of nerve trunks in the adult, comes to the conclusion that, though nerves are made up of anastomosing bundles of fibers, the actual course of the fibers is for the most part a straight one from the plexus to the point where a peripheral branch is given off. Further, he thought that a given segmental level of the cord is represented by a certain area of the cross section of a nerve trunk if the nerve arises from more than one segment of the cord. A few years later, Stoffel,[2] apparently without knowledge of Bardeen's observations, developed a similar idea of functional topography in peripheral nerves, basing his conclusions on gross dissection and a study of cross sections of nerves at different levels. He diverges from Bardeen in that he considers fasciculi simply as continuations of the peripheral branches of nerves and therefore thinks it possible grossly to recognize high up in a nerve trunk the exact fasciculi or groups of fasciculi which make up a given peripheral branch. He figures cross sections of nerve trunks showing the arrangement of the bundles according to the branches which he thinks they represent. With this idea in mind he sets down various rules of operative procedure to be followed in the case of nerve suture, transplantation and the like.

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* A thesis submitted to the faculty of the graduate school of the University of Minnesota, in partial fulfilment of the requirements for the degree of Doctor of Philosophy in Nervous and Mental Diseases, 1921.

1. Bardeen, C. R.: Development and Variation of the Nerves and the Musculature of the Inferior Extremity and of the Neighboring Regions of the Trunk in Man, Am. J. Anat. **6**:259, 1906.

2. Stoffel, A.: Beiträge zu einer rationellen Nervenchirurgie, München. med. Wchnschr. **60**:175 (Jan.) 1913. Neues über das Wesen der Ischias und neue Wege für die operative Behandlung des Leidens, München. med. Wchnschr. **60**:1365 (June) 1913. Stoffel, A. und Vulpius, O.: Orthopädische Operationslehre, Stuttgart, 1913. Stoffel, A.: Ueber die Behandlung verletzter Nerven im Kriege, München. med. Wchnschr. **62**:201 (Feb.) 1915.

General interest in this field was lacking, however, until the great war centered the attention of physicians on nerve anatomy and physiology with the hope that nerve therapeutics would be furthered by such new facts as could be ascertained.

A new method investigating this problem was introduced during the period of the war by Marie.[3] By faradic stimulation of nerves exposed at operation and noting the consequent muscular contraction, this writer [4] reached essentially the same conclusions as Stoffel. Other workers [5] have taken up and confirmed many of Marie's findings. The ideas of Stoffel, Marie and their supporters have received general recognition and are much quoted in important treatises on the diagnosis and treatment of nerve lesions.[6]

But this conception of tract systems within peripheral nerves, especially of fasciculi or groups of fasciculi representing peripheral branches of the nerves, has not gone unchallenged. Sherren [7] states that:

> Certainly a third of the (nerve) trunk, may be divided without producing motor or sensory change or one of transient nature only. I have on several occasions divided one third of the internal popliteal nerve without producing any paralysis, often no sensory change, occasionally a loss of power of appreciating light touches which returned in a few days.
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> * * *
>
> But if the incision cuts into the nerve just above the point of origin of a branch the signs of complete division of that branch are produced. In certain situations also—for example, in the anterior primary division of the fifth cervical nerve—the nerve fibers are arranged in a well-defined order, and incomplete division of this nerve may entail complete division of those motor fibers which supply the spinati and the deltoid muscles. Again, in the trunk

3. Marie, P.: Des résultats fournis par l'électrisation directe des troncs nerveux dans la plaie opératoire chez les blessés atteints de traumatismes des nerfs, Bull. de l'Acad. de méd. **73**:173 (Feb.) 1915.

4. Marie, P.; Meige, H., and Gosset, A.: Les localisations motrices dans les nerfs périphériques, Bull. de l'Acad. de méd. **74**:798 (Dec.) 1915.

5. Putti, V.: Sulla topografia fascicolare dei nervi periferici e più specialmente dello sciatico popliteo esterno, Clin. chir. **24**:1021, 1916. Benisty, Mme. A. (Translation by Buzzard, E. F.): Treatment and Repair of Nerve Lesions, University of London Press, London, 1918. Kraus, W. M., and Ingham, S. D.: Electrical Stimulation of Peripheral Nerves Exposed at Operation, J. A. M. A. **74**:586 (Feb. 28) 1920. Peripheral Nerve Topography—Seventy-Seven Observations of Electrical Stimulation of Normal and Diseased Peripheral Nerves, Arch. Neurol. & Psychiat. **4**:259 (Sept.) 1920.

6. Tinel, J. (Translation by Joll, C. A.): Nerve Wounds, William Wood & Co., New York, 1918. U. S. Army Manual of Neuro-Surgery, Government Printing Office, Washington, 1919.

7. Sherren, J.: Injuries of Nerves and Their Treatment, William Wood & Co., New York, 1907.

of the great sciatic nerve, the external and internal popliteal nerves remain separate from their origin in the pelvis, hence incomplete division of the great sciatic may cause complete division of the external or internal popliteal nerve.

Heinemann [8] has made dissections of the human median, ulnar and sciatic nerves, following the methods of Stoffel.[2] He concludes that Stoffel's conception is entirely erroneous and misleading. The figures of his dissections show plainly numerous and irregular anastomoses and divisions of the bundles of these nerve trunks throughout their whole lengths.

Langley and Hashimoto,[9] without reference to Stoffel's or Heinemann's work, have made dissections similar to theirs, using the sciatic trunk, the tibial and common peroneal nerves of man and of some of the domestic animals. They describe close plexuses along the course of these nerves and are unable to trace muscular branches proximally, as entities, for any great distance, though certain cutaneous branches remain separate for relatively long distances (24 cm. in the case of the N. cutaneous surae lateralis).

Dustin [10] has made an extensive study of the median, ulnar and radial nerves. He studied them in a way similar to those of Heinemann, and Langley and Hashimoto. In addition he made cross-sections at centimeter intervals so that he could follow their fascicular topography at different levels more accurately than had been done in the past. Kraus and Ingham [5] are the only writers on the subject who have referred to Dustin's work. They state that "the work is purely anatomical and, though of importance, led to no correlation with the functions of the fasciculi he describes." This is true but misleading, as can be seen from Dustin's article:

1. The arrangement of fasciculi varies considerably from nerve to nerve and from individual to individual.

2. The fascicular topography of the same nerve is continually modified by the exchange of numerous anastomoses between the various fasciculi.

From the above it becomes impossible to conceive of a functional systematization of nerves with fasciculi as its basis anatomically. However, one may admit that in every quadrant of a nerve (anterior, posterior, internal and external) such and such motor or sensory fibers are grouped by preference.

8. Heinemann, O.: Ueber Schussverletzungen der peripheren Nerven. Nebst anatomischen Untersuchungen über den inneren Bau der grossen Nervenstämme, Arch. f. klin. Chir. **108**:107 (Aug.) 1916.

9. Langley, J. N., and Hashimoto, M.: On the Suture of Separate Nerve Bundles in a Nerve Trunk and on Internal Nerve Plexuses, J. Physiol. **51**:318 (Sept.) 1917.

10. Dustin, A. P.: La fasciculation des Nerfs, Ambulance de "l'Ocean" **2**:135 (July) 1918.

One may not, however, subscribe to the exact ideas of Stoffel nor to the conclusions of P. Marie, Gosset and Meige on the existence of constant, distinct fasciculi definitely situated in the nerve trunks.

Results obtained by localized electric simulation are of value only for a given nerve at a given level.[11]

FASCICULI IN THE HUMAN SCIATIC

The right sciatic nerve of an adult human being was dissected and drawn natural size on a piece of plotting paper. Fig. 1 represents this plotting reduced to one-quarter its natural size. The nerve was cut into blocks, 1 cm. in length; these were numbered serially and oriented spatially by stringing them on a thread which passed posteroanteriorly through the proximal end of each. They were imbedded in paraffin and cut serially, still keeping them oriented in space. For the most part sections were mounted at intervals of 3 mm., though two blocks were mounted entire in order to trace the actual anastomoses and divisions of the fasciculi within a limited extent of the nerve (Fig. 6). The sections were stained with hematoxylin and orange G.

The numbers in Figure 1 refer to sections shown in Figures 2 to 5. For example, in Figure 1, Numbers 10, 10′, 10″ indicate the levels of the sections diagrammed in Figure 2, number 10, Figure 4, number 10′ and Figure 5, number 10″, respectively. Figure 6 is drawn from sections taken from above the level at 10 to below that at 11 in Figure 1. Figures 2 to 5 are projectoscopic drawings. The fasciculi are shown in solid black and the connective tissue which cuts the fasciculi into groups is shown by lines.

Examination of Figures 2 and 3, which represent sections at centimeter intervals from the sacral plexus to the bifurcation of the sciatic, shows that no two sections have a similar fascicular topography. There is variation in the number, position and size of the bundles from section to section. Furthermore, the grouping of the fasciculi is variable from section to section in practically every case. The number of fasciculi is variable in the different sections, but in general they become larger and less numerous as the knee is approached, owing evidently to more fusion than splitting. If one then traces the sections out into the common peroneal and tibial it is seen that this fusion continues so that in the common peroneal (Fig. 5, section 5″) there is a single bundle except for three small ones which are about to form the N. cutaneous surae lateralis and a small cutaneous ramus to the skin over the capitulum fibulae. In the tibial the reduction in number of the fasciculi is also marked; there are only three in the tibial trunk proper at the

11. Dustin's article was not available to me until my investigations had been completed (March, 1921). It was at once a disappointment and a satisfaction to note the close correspondence of our observations.

level of Figure 4, Section 8′. If the sections are traced still farther, the fasciculi are seen to redivide into a considerable number of smaller bundles, apparently in preparation for the sending off of branches along the more distal course of the nerves.

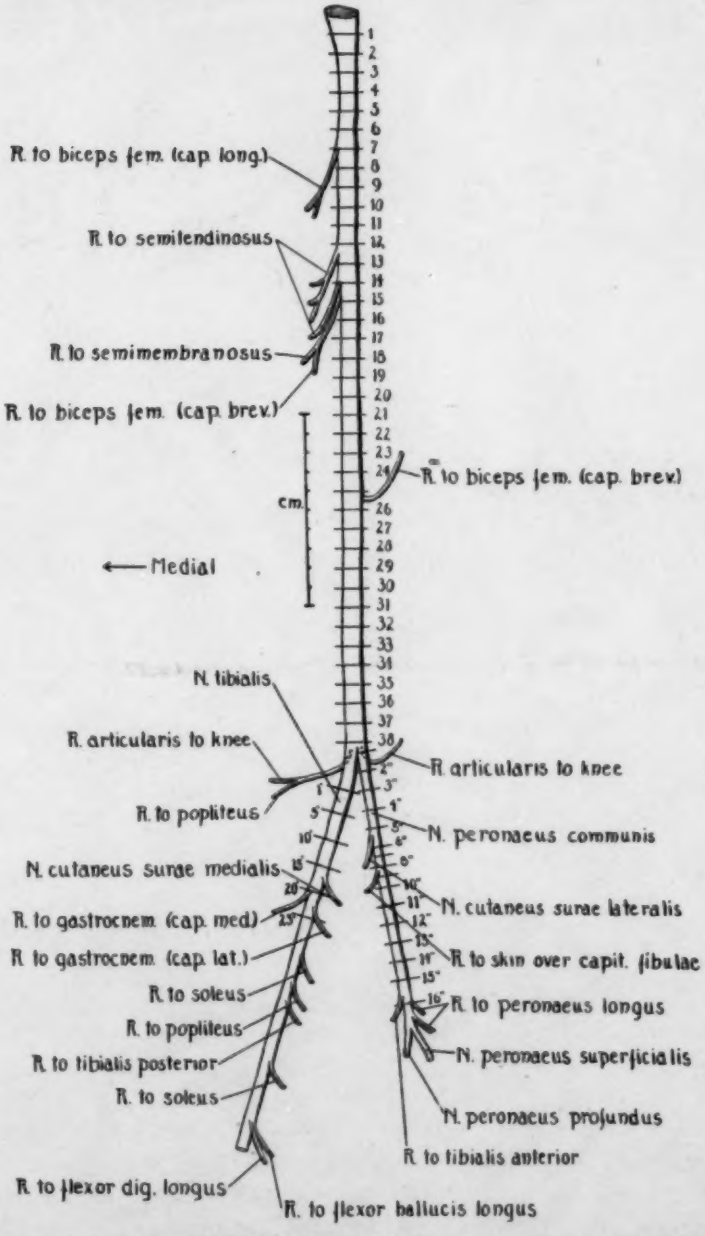


Fig. 1.—Diagram of the right sciatic nerve of an adult human being. The levels of the sections in Figures 2 to 6 are indicated by corresponding numbers in this figure, one-fourth natural size.

The behavior of the fasciculi as they go to form the branches of the sciatic is of interest. While it is probably true that the strands making up the common peroneal nerve do not fuse with any of the tibial nerve bundles and vice versa (we shall return to this in the next section of the article), it is possible to trace the respective groups only about half way up the sciatic trunk before we become uncertain as to

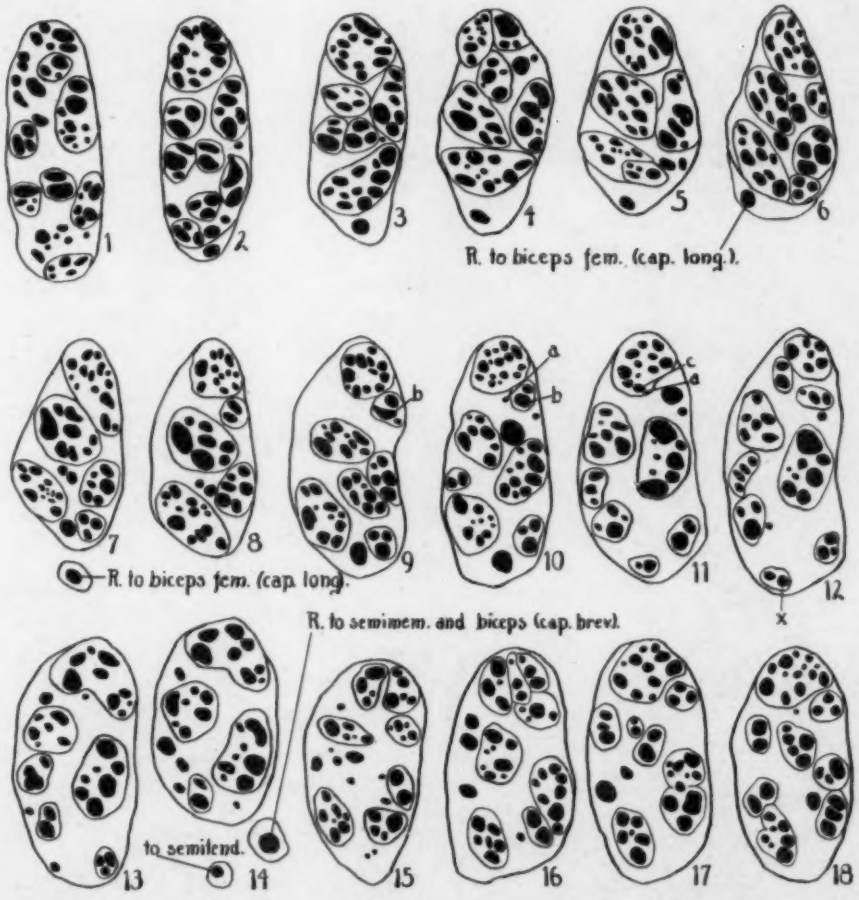


Fig. 2.—Sections at centimeter intervals. Fasciculi are shown in solid black. The connective tissue grouping of the bundles is indicated by the lines. Lateral is to the top of the page, posterior to the right; X 3.

whether there is crossing over of the fibers. For example, in Figure 3 at number 38 the peroneal and tibial groups are widely separated; at number 19 or 20 the separation is questionable; and in Figure 2 it becomes practically impossible without sections at closer intervals to follow the two groups as separate entities.

In the case of the fasciculi forming the collateral branches of the sciatic, the tibial and the common peroneal nerves, it will suffice to mention only those branches which are figured as they will serve to indicate the general principles of formation of collateral branches.

In Figure 2, section 7, the branch to the long head of the biceps femoris is seen to be leaving the sciatic. The fasciculus of this branch

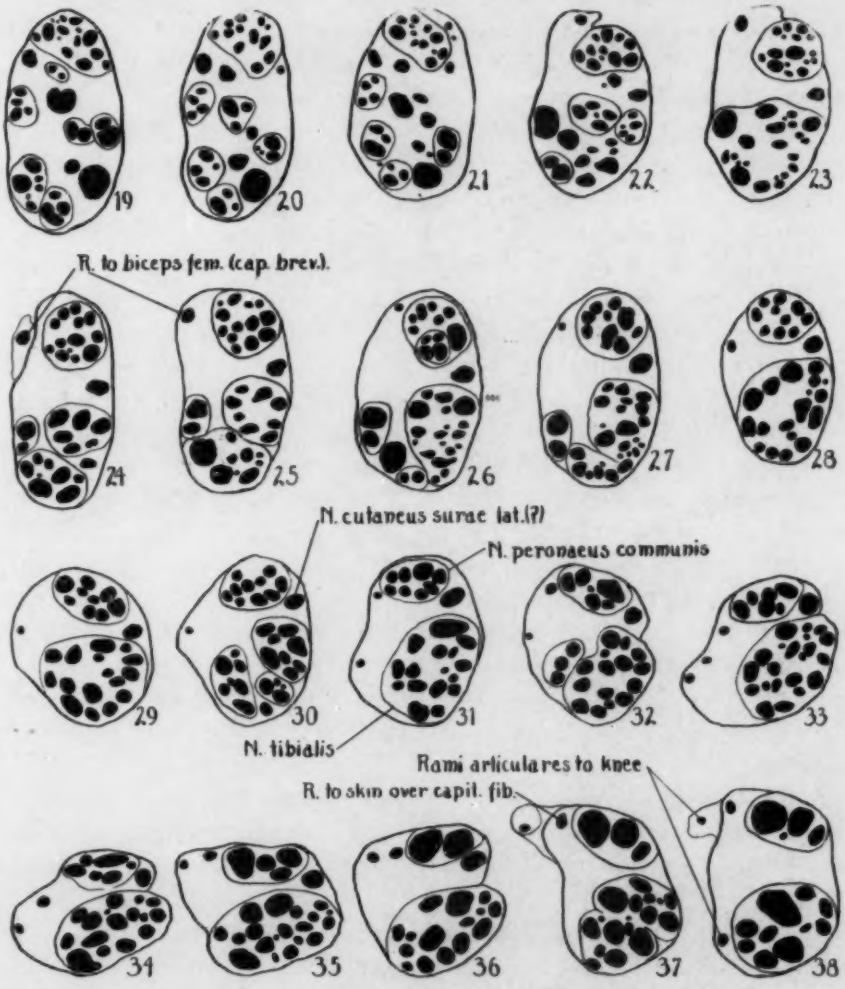


Fig. 3.—Continuation of Figure 2.

can be successfully traced to section 2, or a distance of about 6 cm. Between sections 12 and 13 a small branch is given off to the semitendinosus (Fig. 2, section 12 x) which is closely related to another branch to the semitendinosus which is seen leaving the sciatic in section 14. These two branches form fasciculi which fuse when traced proxi-

mally into one bundle (section 10) which still farther proximally is again broken up (section 8). Thus the fasciculi to the semitendinosus are traceable as anatomically separate pathways for not over 5 cm. along the nerve trunk from the point of departure of the branch. The other collateral branch leaving the sciatic trunk in section 14 runs to the semimembranosus and the short head of the biceps. The fasciculi to this branch can be traced proximally no farther than section 9 (5 cm.). Just distal to section 25 a branch is given off to the short head of the biceps, the fasciculus of which is traceable into section 22 (about 4 cm.). It is conceivable that this bundle could be traced above this point if we had serial sections, but certainly for not more than 4 or 5 cm. additional. Immediately below section 38 two small rami articulares to the knee joint leave the sciatic trunk, one on the tibial and one on the common peroneal side of the nerve. The one on the tibial side is almost immediately lost among the other tibial bundles, but the one on the peroneal side can be traced as high as section 26 (12 + cm.).

Three branches from the tibial trunk are shown in Figure 4. The sections are approximately 3 mm. apart. The N. cutaneous surae medialis is given off immediately below section 17′ (not shown in sections 16′ and 17′ in order to save space on the drawing) and can be traced proximally as a single fasciculus to section 1′ (but not to section 38), about 6 cm. above the point of departure from the tibial. Just below the level of section 20′ a branch is given off to the medial head of the gastrocnemius (see section 13′ for its identification and compare with Figure 1) the fasciculus of which can be traced to section 1′ but not to section 38. The extent of this fasciculus as a separate bundle is thus about 7 cm. The branch to the lateral head of the gastrocnemius leaves the tibial trunk at the level of section 22′ (see section 11′ for its identification) and can be traced also to section 1′, but not higher, a distance of about 7.5 cm.

In Figure 5 are shown sections from the common peroneal trunk at the levels indicated in Figure 1. The N. cutaneus surae lateralis leaves the common peroneal just below section 5″ and is traceable with certainty as a single fasciculus into section 3″, though from the figures it is possible that this fasciculus may extend as high as section 22, a distance of about 21 cm. It is evident that these fibers are located within a definite area of the nerve, while it must be admitted that other fibers probably run along with them (Fig. 3, section 30—N. cutaneous surae lateralis?). The branch to the skin over the head of the fibula leaves the common peroneal nerve near the level of section 8″—the fasciculi can be seen in section 7″. This branch is traceable with fair certainty to section 32, a distance of 13 cm. The branch to the tibialis anterior leaves the common peroneal about 0.75 cm. below section 15″ and

cannot be identified in section 14″ so that its fasciculus remains separate from the rest of the nerve for a distance of not over 2 cm. Three small rami to the peroneus longus are seen to be leaving the peroneal trunk at and immediately below section 16″. These branches are represented by three small fasciculi in section 16″ which cannot be

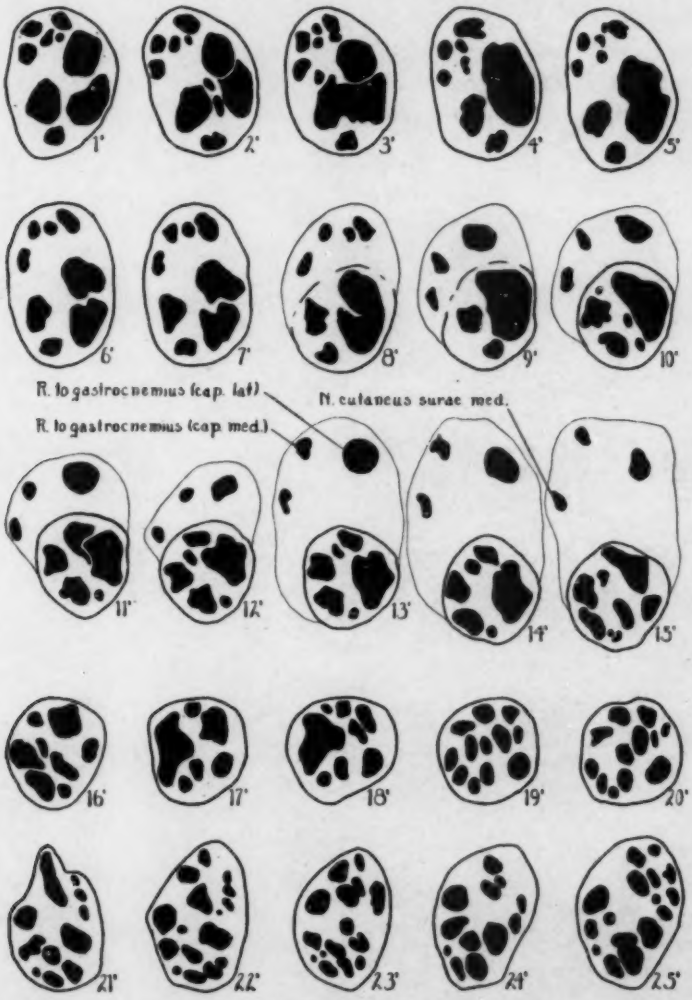


Fig. 4.—Sections at 3 mm. intervals of the right tibial nerve. The numbers of the sections correspond to those in Figure 1. Fasciculi are shown in solid black. Lateral is to the top of the page, posterior to the right; X 4.

identified above section 15″, a distance of about 1.5 cm. In this same section (16″) we note that the common peroneal is dividing into its two terminal branches, the superficial and the deep peroneal nerves. The two corresponding groups of fasciculi apparently remain separate from one another up to sections 8″ or 9″, a distance of about 5 cm.

It also seemed worth while to determine accurately by serial sections, the exact amount of fusion and division of the fasciculi within a limited extent of the sciatic at a point where the sectional topography does not indicate a rapid shifting of the nerve fibers from one bundle to another. Two centimeter blocks, 10 and 11 (Fig. 1), were chosen for this purpose. Figure 6 is a graphic representation of the results of the study of these two blocks. The sections drawn were taken at sufficient intervals so that a number of anastomoses or divisions of the fasciculi have occurred between any successive two of them. Fasci-

Fig. 5.—Sections of the right common peroneal nerve. The numbers of the sections correspond to those in Figure 1. Fasciculi are shown in solid black. The connective tissue grouping of the fasciculi is indicated by the lines. Lateral is to the top of the page, posterior to the right; X 4.

culi which do not fuse or divide in the course of the nerve between any two of the sections shown are drawn in stipple; fasciculi which do divide or fuse are shown in solid black. Section 1 contains forty-nine fasciculi and section 8 contains forty-one, a reduction of eight bundles within these two centimeters. This reduction would be most simply explained by assuming that eight fusions of the fasciculi had occurred in the region. However, we find that from section 1 to

section 8 there are twenty-three fusions and fifteen divisions, a predominance of eight fusions. Thus, in this limited extent there is considerable passage of fibers from one bundle to another, though on superficial examination it would appear that the fascicular topography did not vary to any marked degree. Another point of interest is indicated in this drawing. The fasciculus marked "a" is an offspring of the one marked "b" (compare with Figure 2). Within these two

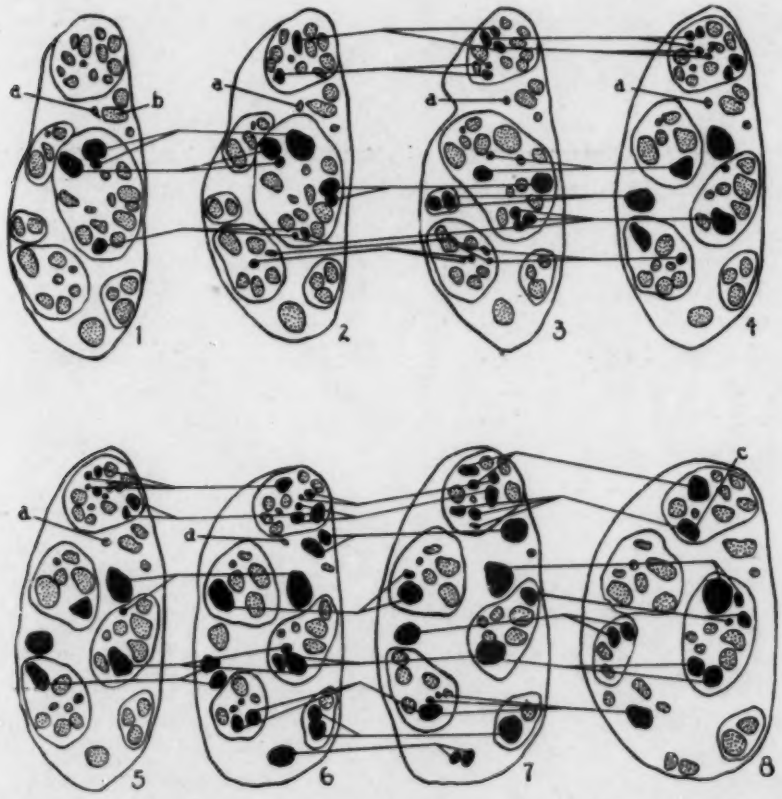


Fig. 6.—Anastomoses and divisions of fasciculi in 2 cm. of the sciatic trunk from above section 10 to below section 11 in Figure 1. Anastomosing or dividing fasciculi are shown in solid black; the others are stippled. Lateral is to the top of the page, posterior to the right; X 4.

centimeters "a" moves from the more posterior portion of the nerve, crosses the midline and comes to lie in the more anterior portion, after which it is incorporated in the fasciculus "c". This shows that groups of fibers may quickly cross from one side of the nerve to the other without any marked change in the topographical picture, though as a rule the anastomoses appear to concern adjoining fasciculi.

The foregoing findings may be summarized as follows:

1. The sciatic is made up of fasciculi which fuse and divide throughout the course of the nerve, forming a vast intraneural plexus.

2. Fusion predominates over division proceeding distally, so that proximally the sciatic is made up of a large number of relatively small fasciculi while in the more proximal portions of its two terminal branches the nerve is composed of relatively few large fasciculi. From about the level of the knee distally, the reverse holds true. The proximal part of the tibial and common peroneal is composed of few large fasciculi which soon split up into a large number of small ones as the more peripheral branches are formed.

3. There is continued crossing of fibers from one fasciculus to another, consequently opportunity for a complete change of functional topography throughout the course of the nerve (e.g., "a" in Fig. 6).

4. Fasciculi in the trunks of the common peroneal, tibial and sciatic nerves may be traced proximally from their respective points of departure for variable distances up the nerve trunks. Aside from the separation of the sciatic trunk into the tibial and common peroneal (which is probably complete), the bundles to the various collateral and terminal branches can be traced a maximum of 21 cm., a minimum of 1 cm. and an average of 6.5 cm. before they are lost in the neighboring fasciculi.

THE COURSE OF DEGENERATED FIBERS IN PARTIALLY DIVIDED NERVES

In order to study the course of the fibers within the sciatic, the following method was undertaken on a number of dogs:

The nerve was bared under aseptic conditions between the ischial tuberosity and the greater trochanter of the femur and was partially divided with a very fine cataract knife so as to cut through various segments of the nerve in the different experiments. The dogs were killed after fourteen days and the sciatic nerves with their branches were stained according to the Marchi method. We could thus study the course of the fibers in a given segment of the nerve just as tracts in the spinal cord have been studied.[12]

Figures 7 and 8 graphically tabulate the results.

Figure 7 shows the degenerations obtained from division of the common peroneal at the level stated. In the figures the degenerated areas of the nerve are shown in stipple, while the normal regions are

12. In these experiments free use has been made of Ellenberger, W. u. Baum, H.: Anatomie des Hundes, Verlag von P. Parey, Berlin, 1891. Serial sections in the longitudinal plane were made to determine the presence or absence of degenerated fibers throughout these experiments.

	Exp. 1.	Exp. 2.	Exp. 3.	Exp. 4.	Exp. 5.
N. ischiadicus					
R. to biceps femoris	None	None	None	Complete	None
N. peronaeus commun.					
N. peronaeus superficialis	Complete	No sections	Occasional	¼	⅔
N. peronaeus profundus below rami to leg muscles	Complete	⅓	Occasional	Occasional	½
R. to tibialis anterior	Complete	⅓	None	Occasional	⅕
R. to extensor digitorum longus	Complete	½	None	Occasional	¼
R. to peronaeus longus	Complete	⅓	Occasional	None	¼
R. to peronaeus tertius	Complete	⅓	None	Occasional	No sections
R. to peronaeus brevis	Complete	½	Occasional	Occasional	No sections

Fig. 7.—Marchi degeneration following partial division of the common peroneal nerve. The branches under consideration are indicated in the column at the left. Degenerated fibers are shown in stipple; normal areas are left blank. "N. ischiadicus" indicates a section of the nerve just below the level of the lesion. "N. peronaeus communis" indicates a section of the common peroneal at the level of the knee. Anterior is to the left, lateral is to the bottom of the page for the sciatic and common peroneal sections.

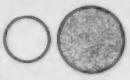
	Exp. 6.	Exp. 7.	Exp. 8.	Exp. 9.
N. ischiadicus				
R. to biceps femoris	None	None	None	Complete
N. tibialis				None
Superficial branch to skin of back of leg	Complete	No sections	¼	None
Deep branch to skin of back of leg and skin and muscles of sole of foot	Complete	No sections	½	None
R. to gastrocnemius (caput mediale)	Complete	No sections	⅓	None
R. to gastrocnemius (caput laterale)	Complete	Occasional	¼	None
R. to flexor digitorum sublimis	Complete	Occasional	Occasional	None
R. to flexor digitorum profundus	Complete	No sections	Occasional	None
R. to tibialis posterior	Complete	Occasional	Occasional	None
R. to popliteus	Complete	No sections	Occasional	None

Fig. 8.—Marchi degeneration following partial section of the tibial nerve. The branches under consideration are indicated in the column at the left. Degenerated fibers are shown in stipple, normal areas are left blank. "N. ischiadicus" indicates a section of the sciatic trunk just below the level of the lesion. "N. tibialis" indicates a section of the tibial trunk at the level of the knee. Anterior is to the left, lateral is to the bottom of the page for the sciatic and tibial nerve sections.

left blank. Thus, in Experiment 1 the whole common peroneal nerve was cut across, and we see a corresponding complete degeneration of all the fibers contained within it. Each of the branches of the common peroneal is likewise totally degenerated. No degeneration was found in the tibial nerve trunk nor in any of its branches.

In Experiment 2 approximately the lateral half of the common peroneal was divided, and we see a corresponding degeneration with the Marchi stain. The heading "N. ischiadicus" indicates a section of the sciatic 1 or 2 cm. below the level of the lesion. At the level of the knee the degenerated fibers are scattered throughout the whole diameter of the peroneal trunk. These diagrams may be checked by the photo-

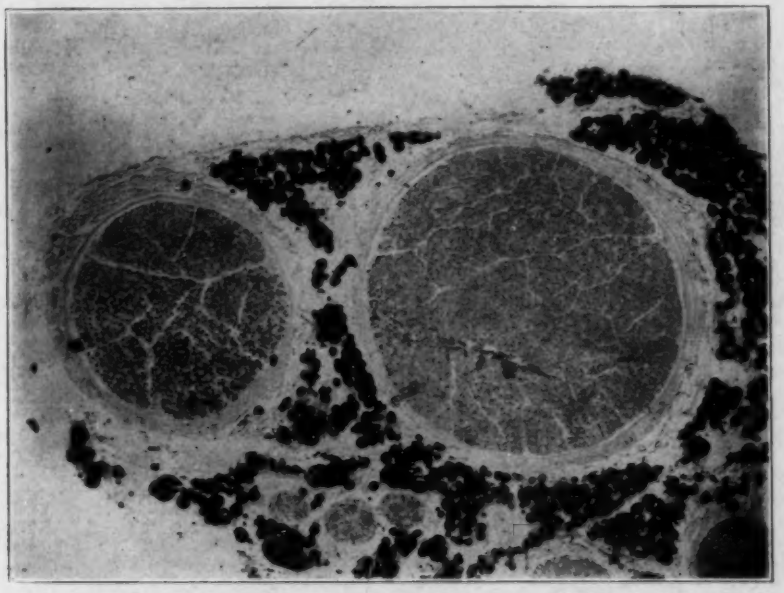


Fig. 9.—Photomicrograph of a Marchi section of the sciatic nerve just below the level of the lesion in Experiment 2. Posterior is to the right, lateral to the bottom of the figure; X 26.

micrographs shown in Figures 9 and 10. Figure 9 corresponds to the drawing in Figure 7, Experiment 2, N. ischiadicus; Figure 10 corresponds to Experiment 2, N. peronaeus communis. There is degeneration in all sectioned branches of the common peroneal. About four fifths of the fibers in the N. peronaeus profundus below the point where the rami to the leg muscles are given off are degenerated, about two thirds of those in the branch to the peronaeus longus, about half the fibers to the extensor digitorum longus and the peronaeus brevis, and one third of those to the tibialis anterior and the peronaeus tertius are degenerated. Figure 11 is a photomicrograph of the branch to the

peronaeus longus and may be used for comparison with the drawing of the degeneration of this branch shown in Experiment 2, N. to peronaeus longus (Fig. 7). In this same experiment there is slight degeneration of the anterior part of the tibial nerve which is traced out in Figure 8.

In Experiment 3 (Fig. 7) only a few fibers of the posterior side of the common peroneal were divided (see under N. ischiadicus). No degeneration is demonstrable in the branches to the biceps femoris, the tibialis anterior, the extensor digitorum longus and the peronaeus tertius. In sections of the N. peronaeus communis the degenerated fibers are considerably scattered throughout the cross section of the nerve. Degeneration is demonstrable in about equal amounts in the N. peronaeus superficialis, the N. peronaeus profundus below the rami to the leg muscles, the ramus to the peronaeus longus and to the peronaeus brevis.

The common peroneal in Experiment 4 shows compact degeneration in the anterior part and a few scattered degenerated fibers along the lateral side of the nerve. Also, a small fasciculus of the sciatic accompanying the common peroneal along its anteromedial side is completely degenerated. The branch to the biceps femoris is completely degenerated and is undoubtedly represented by the small fasciculus just mentioned. The N. peronaeus superficialis is about one quarter degenerated; there is no degeneration in the branch to the peronaeus longus and occasional degenerated fibers are found in each of the other rami studied.

In Experiment 5 the lateral third of the cross section of the common peroneal is degenerated, together with a small accompanying fasciculus which is completely degenerated. No completely degenerated branches are seen in this nerve so it must be assumed either that some branch (probably to the biceps femoris) has been lost in the dissection or that this fasciculus has fused and been incorporated again with the common peroneal. From a study of other sections in this series it is most probable that the former is the correct explanation. The N. peronaeus superficialis is about two thirds degenerated, the N. peronaeus profundus below the points of emission of the branches to the leg muscles about one half degenerated, the branches to the extensor digitorum longus and peronaeus longus each about one quarter, and the branch to the tibialis anterior about one fifth degenerated. No sections of the branches to the peronaeus tertius and brevis are available because of difficulty in dissection.

Figure 8 shows results of total and partial divisions of the tibial trunk. The caption "N. ischiadicus" again indicates a section of the sciatic trunk taken just below the level of the lesion. In Experiment 6

the tibial has been divided completely. We find correspondingly complete degeneration of the tibial trunk and all its branches. There is no demonstrable degeneration in the peroneal division nor in any of its branches.

Experiment 7 is from the same nerve that is shown in Experiment 2, but here the tibial degeneration is considered. We find that the small group of degenerated fibers at the anterior margin of the tibial division at the level of the lesion is scattered over at least the anterior

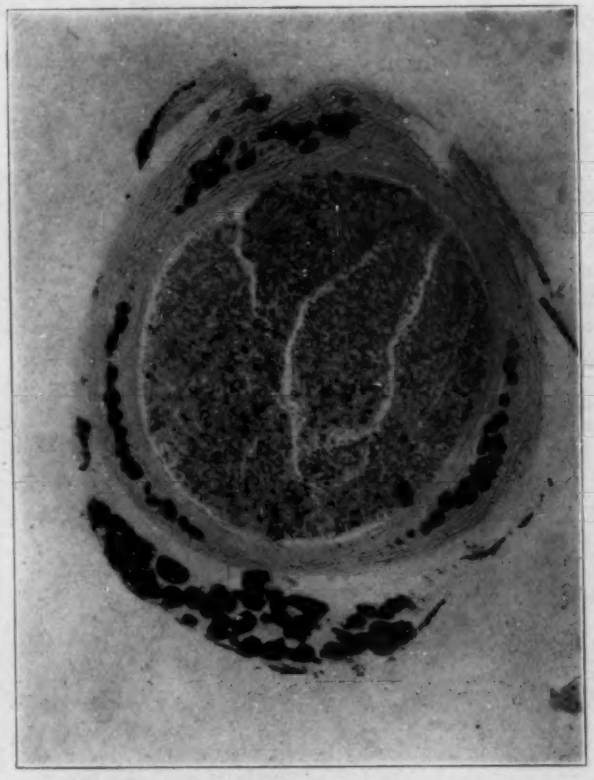


Fig. 10.—Photomicrograph of a Marchi section of the common peroneal nerve at the level of the knee in Experiment 2. Posterior is to the right, lateral to the bottom of the figure; X 46.

half of the tibial trunk when sectioned at the level of the knee. Through some error sections of only three branches of the tibial are available in this experiment. However, all three of these rami show degenerated fibers.

In Experiment 8 the tibial trunk was divided along its lateral, anterior and medial margins (Experiment 8, N. ischiadicus). A small fasciculus accompanying the tibial side of the sciatic was also completely

divided, and we are unable to trace this fasciculus into its terminal branch. The explanation in this case is the same as that already given for an analogous finding in Experiment 5. We see in this experiment (8) that at the knee level the tibial nerve is partially degenerated throughout its whole cross section. Also there is degeneration in every branch of this tibial nerve though most marked in the superficial branch to the skin of the back of the leg, in the ramus to the gastrocnemius and in the deep branch of the skin of the back of the leg and the skin and muscles of the sole of the foot.

In Experiment 9 only a small fasciculus was divided. The only branch of the sciatic found degenerated was a small one to the biceps femoris.

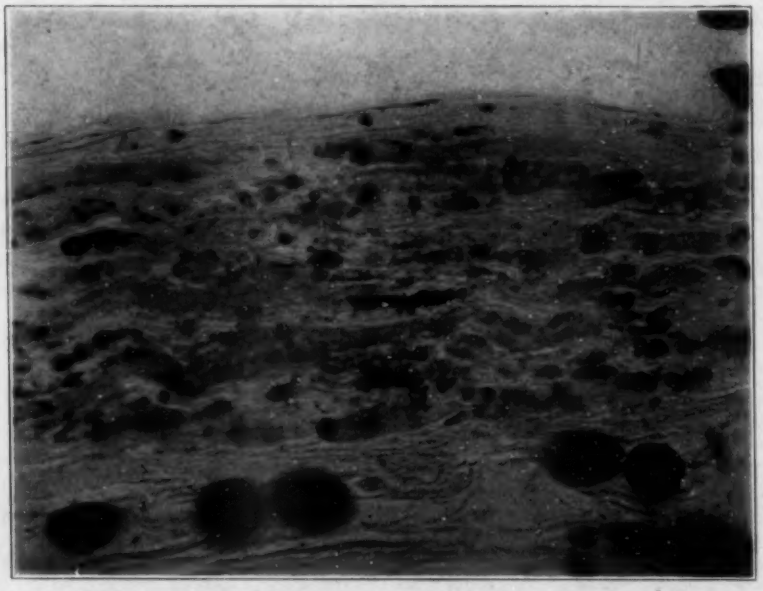


Fig. 11.—Photomicrograph of a longitudinal section of the nerve to the peronaeus longus in Experiment 2. The figure shows the typical appearance of degenerating myelin sheaths in longitudinal section when stained according to Marchi's method. About two-thirds of the fibers are degenerating; X 300.

These experiments show that in the dog the tibial and common peroneal are completely separate from one another at least as high as the level of the ischial tuberosity and the greater trochanter of the femur. Also, partial division of the common peroneal or tibial gives regularly a degeneration which spreads out over most of the cross section of the nerve in its course down the sciatic trunk and is demonstrable in all of the peripheral branches if more than a very small area is divided. The fasciculi to the hamstring muscles are given off for the most

part above the level at which these experiments were performed though occasionally a small fasciculus to the biceps femoris is seen coursing along with, but remaining separate from, the sciatic trunk for a short distance.

It is regrettable that the foregoing observations are so few and that they have been made on the dog, in which the tibial and common peroneal divisions consist each of a single bundle. Probably more satisfactory results would have been obtained had the goat been used, whose sciatic morphologically more closely resembles that of man and in which partial division of the nerve would have been simpler because of numerous fasciculi more or less visible to the naked eye (Langley and Hashimoto [9]).

ELECTRIC STIMULATION OF THE SCIATIC TRUNK IN THE DOG

A single experiment was done. In an anesthetized dog, the tendons around the ankle were dissected out and each connected by means of a string to its respective lever playing on a drum of smoked paper. The sciatic was isolated between the ischial tuberosity and the greater

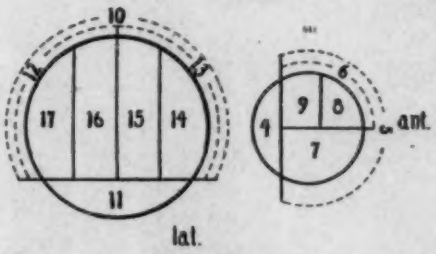


Fig. 12.—Diagram of the segments into which the sciatic nerve was split in the electric stimulation experiment. The numbers of the segments correspond to the tracing numbers in Figures 13 and 14; for example, when segment 4 (above) was stimulated, the record of the muscles which contracted is that in Figure 13, tracing No. 4.

trochanter of the femur, from 3 to 4 cm. below the sacral plexus, was cut across and stimulated by means of an ordinary induction coil with small electrodes. The current used was the minimal to produce contraction of the muscles. In Figure 13, No. 1, is shown the tracing obtained on passing a current through the whole cross section of the nerve. All the muscles of the leg contracted.

With a fine cataract knife the nerve was then split for about 1.5 cm. into its two divisions which were grossly visible: the common peroneal and the tibial. The first was then stimulated. The tracing obtained is shown in Figure 13, No. 2. The muscles supplied by the common peroneal contracted while those supplied by the tibial did not. Then the tibial was stimulated. The four tibial muscles contracted; the peroneal group did not.

Next the common peroneal division was split longitudinally for about 1.5 cm. into a number of segments as diagrammed in Figure 12. When segment No. 4 was stimulated all the muscles of the peroneal group contracted; those supplied by the tibial did not (Fig. 13, No. 4). When the remaining portion of the common peroneal was stimulated (Fig. 12, No. 5) all the muscles of the peroneal group again gave a curve, while those of the tibial remained unaffected (Fig. 13, No. 5). The same was true for stimulation of all the other segments into which the common peroneal nerve was divided. This is easily seen by com-

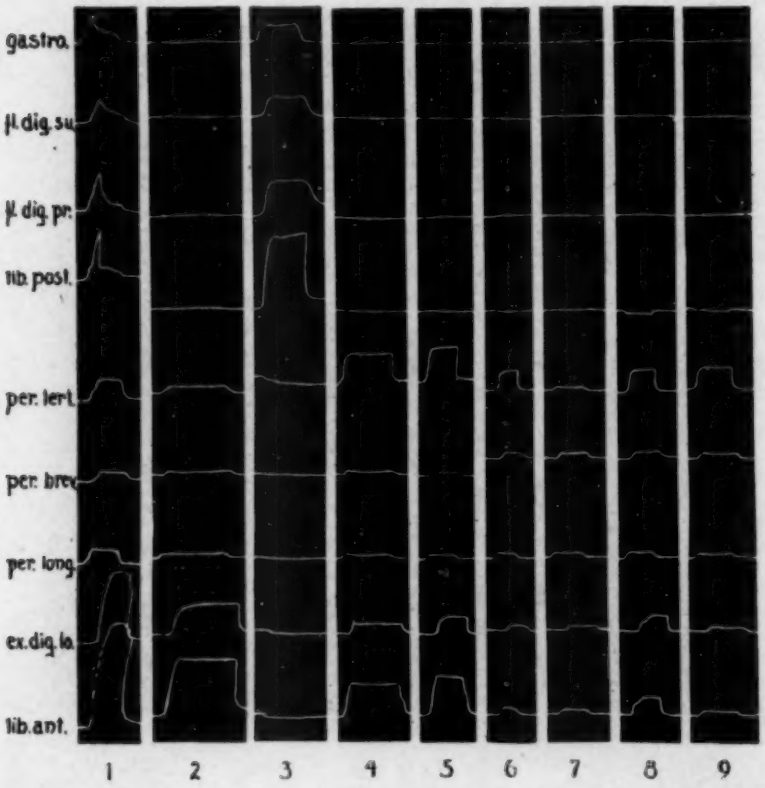


Fig. 13.—Tracings of the muscle contractions in the electric stimulation experiment.

paring the segment number in Figure 12 with the corresponding tracing in Figure 13.

The tibial division was treated in the same way. Here also the tracing number in Figure 14 corresponds to the segment number in Figure 12, which shows diagrammatically the approximate segments into which the tibial was split and then stimulated. In no instance was there contraction of the muscles supplied by the common peroneal.

In every case, except in the stimulation of segments 11 and 14, there was contraction of all four of the muscles of the tibial group from which tracings were obtained. On stimulation of segments 11 and 14 only the flexor digitorum profundus and the tibialis posterior contracted.

We repeat that the current used was the minimal to produce muscle contraction in order to avoid overflow.

This experiment indicates that in the sciatic of the dog at the level between the ischial tuberosity and the greater trochanter of the femur there is little grouping of the motor fibers into functional groups aside from the separation of the nerve into its two main divisions. The

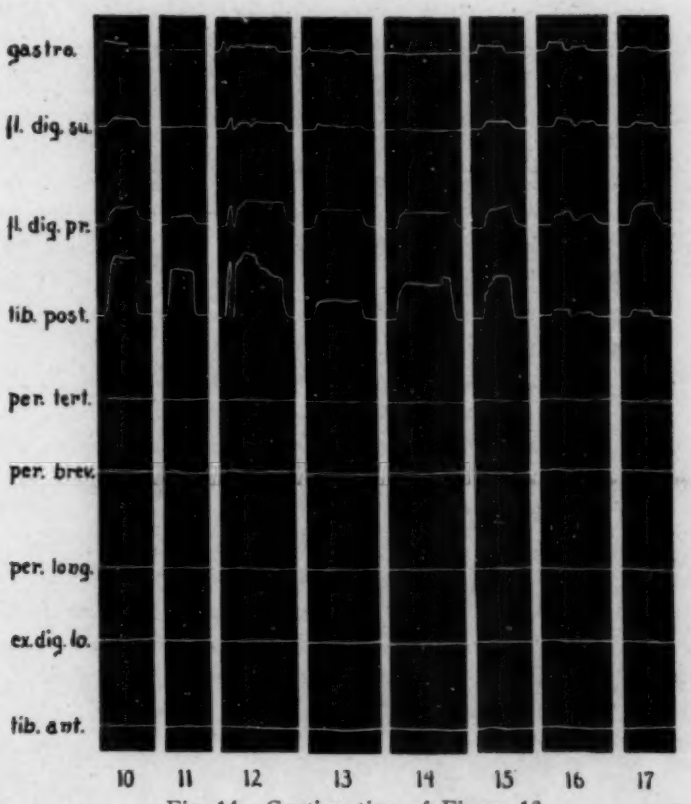


Fig. 14.—Continuation of Figure 13.

observations would have been more satisfactory if they had been carried out on a species whose sciatic is made up of a number of grossly visible fasciculi, as in the human being.

DISCUSSION

It has been shown in the foregoing that the tibial and common peroneal divisions of the human sciatic nerve should be considered as vast plexuses of nerve fibers from the sacral plexus to at least the

distal part of the proximal third of the leg (well into the common peroneal and tibial trunks proper). This continuous exchange of fibers among the various fasciculi making up the nerve can mean only a continuous modification of the functional topography of the nerve throughout its length and definitely negatives the conception of Stoffel,[2] Marie,[3] and Kraus and Ingham,[5] who would have us think of fasciculi in terms of peripheral branches. The only true functional localization in the sciatic, aside from separation into the tibial and common peroneal divisions, is that seen just above the points of departure of branches, since the fasciculi to the branches may be traced along the nerve trunk for an average distance of 6.5 cm. (maximum 21 cm., minimum 1 cm.) before they are lost among neighboring bundles of nerve fibers.

In conformity with this we find, in the dog, that partial division of the sciatic between the ischial tuberosity and the greater trochanter of the femur shows complete separation of the nerve into its tibial and common peroneal divisions, but that partial division of either one of these two causes degeneration, not in single branches but in many of the respective branches of each of them. Hence, there must be very definite mixing of the nerve fibers throughout their course from the sacral plexus and the tibial and common peroneal nerves before peripheral branches are given off.

Electrical stimulation of various sections of the sciatic in the dog at the before mentioned level provokes contraction in all the muscles supplied by either the tibial or common peroneal, depending on which one is stimulated, and not in one or two of the muscles (exception noted in the foregoing).

Thus, in the light of this investigation and that of Dustin,[10] it is impossible to conceive of any functional significance of fasciculi in peripheral nerves in the sense that "the course (of motor fasciculi) is a straight one from the point where the nerve has been made up by its contributing segments to the point of offset of the fasciculi as a branch"—Kraus and Ingham.[5]

In case of a transverse lesion of a nerve it is customary to operate as soon as one is convinced that there is no infection in the field of operation and no evidence of regeneration of the nerve—U. S. Army Manual.[6] The operation then consists in resecting the scar tissue, the neuroma of the central stump and the glioma of the peripheral stump, until the normal nerve fasciculi in the one and the atrophied bundles of the other are distinctly evident. In the average operation, two or three or more centimeters of the nerve must be resected. Then it is advised to suture the divided ends without torsion in as nearly their normal anatomic relationships as possible. Marie says: "It is indispensable that in their sutures, the surgeons appose as exactly as pos-

sible the sectioned ends of the various divided nerve fasciculi of the superior end with the same fasciculi of the inferior end." The impossibility of following to the letter any such procedure is at once apparent. Reference to Figure 6 shows that within 2 cm. of the length of the sciatic trunk there are twenty-three fusions and fifteen divisions of the various bundles at this point and a reduction of eight fasciculi. Thus, after resection of the glioma and neuroma, the fasciculi in the proximal stump cannot correspond to those in the peripheral stump. This, however, does not mean that we cannot support the point of view of a minimum disturbance of the nerve topography in the way of torsion.

As Dustin [10] points out, success of the suture of a nerve depends to a decided extent on the number of fasciculi in the nerve at the point at which it has been divided. If the area involved is where the nerve consists of many bundles, the growth of fibrous connective tissue from the perineurium between the two stumps is much more likely, apposition of normal fasciculi in the proximal stump to those in the peripheral stump becomes much more a matter of chance, and the regenerating axons are much more likely to go astray than if the section has occurred through a "nodal zone" (Dustin) where the fasciculi are few and relatively large, and where there is a minimum amount of connective tissue within the nerve proper. Though no statistics are available, one can deduce from the foregoing that, other things being equal, the prognosis of a sciatic lesion producing complete anatomic interruption is better in direct proportion as the lesion approaches the level of the knee where the fasciculi are few and large.

Again, on the basis of anatomic considerations, it is evident that partial division of the sciatic nerve just above the point of departure of a peripheral branch would be more likely to give symptoms of division of the branch than if the lesion were located higher up; in other words, there is localization of function anatomically for the various branches for only a few centimeters proximal to the level at which they leave the nerve. Hence, partial division of the nerve trunk would probably be more serious as regards a particular branch in direct proportion as the level of the lesion approaches the point of departure of the branch. Sherren [7] has already noted this principle as a clinical fact without explaining the underlying reasons for it.

HEMIHYPERTROPHY AND MENTAL DEFECT

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Anomalies of anatomic development and mental defect are often found in close association. A noteworthy example of this fact came to the writer's attention in 1913. A boy, then 13 years of age, was presented for mental examination. He was found to be mentally deficient. A physical inspection of the whole body of the boy showed this deficiency to be associated with one of the most remarkable of developmental anomalies; namely, total unilateral hypertrophy.

This anomaly is considered to be one of the rarest in the domain of medicine. Marked cases are indeed extremely few as a survey of the literature will reveal; but how frequent mild, unrecognized cases are, remains to be determined.

It is because hemihypertrophy presents a startling, though legitimate, deviation from an almost universal law of development that it constitutes a subject rich in suggestion—and perplexity.

A brief description of the physical and mental characteristics of the case here reported will serve as a basis for the discussion of this interesting condition.

REPORT OF AUTHOR'S CASE

Physical Development.—The boy in stature was fairly well developed for his age. At 20 years of age he was 67 inches tall and 137 pounds in weight. His height-weight index at the age of 13 was also normal, being then 87 pounds for 57⅝ inches. He was phlegmatic in his movements and of moderate endurance. He was one of eight children. The brothers and sisters were average or normal, both physically and mentally. His parents were likewise normal and no facts of hereditary significance could be gained.

The birth was natural, without instrumental assistance, and the mother nursed her child into the second year. Dentition apparently was not delayed.

The mother asserted that she had noticed larger development on the right side in early infancy, and there can be no doubt that the condition was congenital. No children's diseases or accidents of significance were to be noted.

The accompanying tables of measures and illustrations indicate the character and degree of the hemihypertrophy. The first table indicates the physical, and incidentally the mental, status of the subject at the age of approximately 13 years. Seven years later, after adolescence had been practically completed, similar measurements were made and these are presented for comparison. They indicate that the condition of hemihypertrophy is, at least in this case, a relatively stationary one, exhibiting neither a progressive nor ameliorative tendency.

TABLE 1.—TABLE OF MEASUREMENTS (AGE 13)

Height, 57⅝ inches.

Sitting height, 29¼ inches.

Weight, 87 lbs. Head: length, 18.2 inches; breadth, 15.4 inches; circumference, 56.5 inches.

Dynamometer: right, 15; left, 11.

Chronologic age 12½ years; mental age 4½ years; intelligence quotient 36.

	Right	Left	Difference
Arm, length	63	61	2
Forearm, length	38	36	2
Forearm, girth	18	17	1
Wrist	14	13	1
Hand	21	18.5	2.5
Middle finger, girth	7	6.5	0.5
Middle finger, length	10	9	1
Metacarpal bone III, length	46 mm	43 mm	3 mm
Capitale bone, length	20 mm	17 mm	3 mm
Leg, (A. s/s/. to ex. mal.)	80	75	5
Leg, perin. to in. mal.	63	61	2
Leg, patellar edge to in. mal.	34	30	4
Thigh girth	39	38	1
Knee girth	30	29	1
Calf girth	29	28	1
Foot, metatarsal bone, length	54 mm	52 mm	2 mm
Metatarsal bone, width of head	18 mm	15 mm	3 mm
Ex. canthus to antetragus	8	7.5	0.5
Int. canthus to nasolabial junction	5.2	4.5	0.7
Ear, length	7.5	6.5	1
Lip, upper	4	3	1
Upper canine (width)	10 mm	9 mm	1 mm

Examination of the blood and urine gave negative results. The red blood cells numbered 5,424,000; hemoglobin, 95 per cent.; white blood cells, 10,000; polymorphic cells, 68 per cent.; large mononuclears, 21 per cent.; small mononuclears, 11 per cent.

TABLE 2.—TABLE OF REMEASUREMENTS (AGE 20)

Height, 67 inches.

Sitting height, 33¾ inches.

Weight, 137 lbs. Head: length, 18.7 inches; breadth, 15.9 inches; circumference 59 inches.

Dynamometer: right 31; left, 22.

Chronologic age, 20 years; mental age, 5 years, 8 months; intelligence quotient, 35.

Anthropometric Measures	Right	Left	Difference
Arm, length	77.5	75	2.5
Forearm, length	46	44.5	2.5
Forearm, girth	25	23	2
Wrist	16.5	14.5	2
Hand	23	20	3
Middle finger, girth	7.5	7	0.5
Middle finger, length	12.5	11.5	1
Cranium: segments of intertragal arc	19	17	2

The axillary temperature was equal on both sides.

The asymmetry was not very marked. It might easily escape observation, though it caused the boy to limp to a moderate extent.

Careful inspection revealed mild gigantism involving apparently all of the right side: the right ear was longer, the right half of the nose larger, the right nares twice that of the left in diameter, the right palpebral fissure was wider; on the same side the cheek and lips were fuller; the arm was longer, the right hand was relatively more enlarged than some of the other structures; the right leg and foot were similarly enlarged; the penis was definitely enlarged to the right of the median line.

The opthalmoscope showed no marked difference in the right fundus, though the veins were fuller. There were no differences in refraction in the two sides.

Throat examination revealed no marked difference in the tonsils. The uvula was bifid; the epiglottis was definitely asymmetrical, but curiously enough, the left side was thicker than the right; the posterior rim was also bulkier on the left side, the whole right of the epiglottis apparently being in the infantile condition.

A cast (Fig. 5) of the upper palate and teeth showed considerable deformity. The placement of the teeth was very defective on the right side giving the gums a thicker appearance on that side. The right upper canine was 10 mm. wide and 9 mm. thick; the left canine was 9 mm. wide and 8 mm. thick. The eruption was further advanced on the left side, which is contrary to rule.

The skin was by no means normal. The peculiarities, presently to be noted, were more pronounced on the right side but were by no means limited to that side. The skin was dry and there was a definite tendency to ichthyosis. Small keratinous patches were present about the elbows and the ankles. Most prominent and characteristic were extensive areas of telangiectatic congestion which gave the skin a marbled, and, in some areas, a decidedly livid aspect; for example, the dorsal surface of the right hand and the lateral surface of the leg. On last examination the right leg had developed marked varicosities (Fig. 4).

On palpation the hypertrophied side had a more doughy feel than the left. This suggested redundancy of the subcutaneous tissue, but the roentgen rays showed that the bones themselves had participated in the hypertrophy. The measurements showed that all the members of the right side were not proportionately hypertrophied.

Mental Development.—The mental development of this boy has shown a consistent retardation dating no doubt from birth. He was over 2 years old when he began to walk; he began to say "mama" and "papa" at the age of 3; but he did not begin to "talk" until about the age of 5. Even now he speaks in phrases and short sentences and has a very childish vocabulary. He entered the first grade of the public schools at the age of 9. He stayed in this grade two years and was gradually promoted into the higher grades out of deference to his physical development. His academic attainments after years of schooling never rose to those of grade 2. During all these years he has been considered a docile, amenable, cheerful and neat boy. He has been able to read a few words, to spell a few others and to repeat a few number combinations. Persistent drill has left some precipitate, but there has been a restricted intellectual development during the seven years of teens in which I have observed him. He is now 20 years old and, although his intelligence is more mature than it was at the age of 12½, he still must be classified as an imbecile.

At the chronologic age of a little less than 13, he had a mental age of 4½ years by the Vineland revision of the Binet tests. At that time he could not tell which was the longer of two lines, one 7, and the other 6 cm. in length; he was also unable to repeat a sentence consisting of ten words. In another year he was able to meet both of these tests, but otherwise he showed little progress. After six more years of growth, he is still unable to distinguish between right and left and cannot tell whether it is morning or afternoon; but he is able to count 13 pennies, and he can describe a simple picture. The net result is that now at the age of 20 he has a mental age of 5 years and 8 months. In other words, it has taken about seven years for this boy to accomplish the equivalent of one year of mental growth. This growth has been as steady and consistent as his physical growth; but it has been slower and on a different level. There has been no deterioration. Seven years ago the ratio between his mental age and chronologic age was 4½ : 12½, which expressed on a percentage basis gives an intelligence index or quotient of 36. By a similar method of calculation, using the Stanford revision of the Binet scale, we find that his present intelligence quotient (IQ) is 35. Though these two quotients were not derived by precisely the same method, they are comparable and true to the facts of the case. We cannot say that this youth has shown no mental growth during his teens; but viewed in relative terms, we are obliged to conclude that there was no increase in his caliber. His IQ has remained fairly constant.

His actual mental status is summarized in the accompanying normgraph (Fig. 8), which in terms of age norms, plots his ratings. In height and weight he was, and is, at standard; but in the psychophysical measurements obtained by the dynamometer and spirometer, and in the mental and educational measures he is at the level of a beginner at school. On this level he will remain. At the age of 20 his drawings of a house, man and bird are characteristic of a kindergarten mentality.

It would be unjust, however, to leave this sketch without referring to the surprising success which this boy has made in life in spite of his limitations. His mother, though a struggling widow with eight children, was loathe to consider commitment to an institution. We respected this attitude and repeated efforts were made to find a simple occupation for him. There were great difficulties as employers would not take the necessary risk, pains and patience. Finally, the cooperation of neighbors was enlisted and he got a job as a scullery boy at a downtown café, with hours from 8 to 5 and wages of $5.00 a week. For a whole year he has been doing regular, satisfactory work, the pride of his mother. And incidentally this is an example of the sort of thing that local communities might deliberately do to control the problem of feeblemindedness.

SURVEY OF RECORDED CASES OF HEMIHYPERTROPHY

Thomas Wedders, with a nose reputed to be 7½ inches long, was a famous character in an earlier day. The scientific literature on the subject of hypertrophy, however, goes back to the publication of a case by Wagner in Germany, in 1839, and one by Desvouges in France in 1856, and by Adams in England in 1858. Osler, writing in 1879, gave the number of cases of congenital hypertrophy reported to date as twelve. Ely Leblanc, who in 1897 wrote his medical thesis on the subject, made an exhaustive canvass of the literature and

Fig. 1.—Hemihypertrophy; subject aged 13.

assembled seventy-nine case, of which twelve were cases of total hemihypertrophy, thirty-eight of partial, nineteen involved but one extremity, six the face and four were crossed. Lewin assembled sixty cases of hypertrophy limited to fingers and toes. David M. Greig, in 1898, in the Edinburgh Hospital Reports reviewed a collection of forty-two cases. This collection was confessedly incomplete and included a large proportion of cases of partial hypertrophy as distinguished from total unilateral hypertrophy or true hemihypertrophy. Greig's valuable monograph deals chiefly with unilateral cephalomegaly (unilateral hypertrophy limited to the head and face), of which he collected thirty-five cases. I am indebted to Greig and Leblanc for references to several cases considered below.

I was able to collect forty cases of true hemihypertrophy, and this may be regarded as a reasonably complete list of the cases now available in the medical literature. Table 3 gives a list of these cases. For comparative purposes a tabulation of thirty cases of partial and crossed hypertrophies is included. Table 4 is by no means complete, for such partial or restricted hypertrophies are much more common than true total hemihypertrophy. It is not unlikely that they frequently have a pathology and etiology identical with that of hemihypertrophy. We shall, however, limit our discussion to the latter condition.

An analysis of Table 3 shows that of forty reported cases only five patients were adults, nineteen were male, seventeen female, and four not specified as to sex. In twenty-seven, or almost 70 per cent. of the cases, the total unilateral hypertrophy was on the right side. For every case of left-sided hemihypertrophy there are two right-sided cases. In fifteen of the cases it was definitely stated that all the tissues were involved in the hypertrophy. In nineteen cases abnormalities of the skin were mentioned as a complication, while in three cases the skin was reported as normal; in the remaining number no facts on this point were given. The data as to mental status are incomplete. In twenty-nine cases no facts are given; in many of the instances, indeed, the subject was too young to make the mental status obvious. In only six cases was the mentality reported as normal and in five cases, or 13 per cent. of the total, it was reported as defective.

A general statistical summary of the facts in Table 4 shows a similar picture. Only three of the listed cases of partial hypertrophy occurred in adults; males were slightly more numerous (fourteen to eleven); the right side was more frequently involved than the left in the ratio of 16:9. In five cases, however, both sides were partially involved in the same person. Skin complications were reported in eleven cases; in only one case were they recorded as absent. Mentality

Fig. 2.—Same subject as in Figure 1, aged 20.

TABLE 3.—CASES OF TOTAL HEMIHYPERTROPHY

Author	Sex	Age	Side	Mentality	Skin Complications	Special Features
Adams, S. S.	M	10	Right	Normal	Telangiectasis	Hair darker and longer on right side
Ahlfeld	F	2	Left			
Arnheim	F	2	Right	.....	Nevuspilosus hypertrichosis	Right teeth adult in size; right kidney 1 cm. longer; tongue asymmetric; necropsy
Bassoe	M	16	Left	.....	Nevi pigmented hypertrichosis	
Broca	M	14	Left	Subnormal		
Brüning	M	1	Right	.....	Nevi pigmented angioma	Hypertrophy of 2 fingers, left side
Bull	F	..	Left			
Burlet	F	27	Right	Subnormal	Vascularity increased	Temperature + on right side
Cagiati	F	15 mo.	Left	.....	Nevi pigmented keratosis	
Demme	F	1 wk.	Left	.....	.....	Necropsy: left cerebral hemisphere +
Desvouges	M	17	Right	.....	Nevi; varices	2 left toes +; right side stronger
Eaton	M	10	Left			
Fatichi	F	53	Left			
Finlayson	F	18 mo.	Right	.....	Dark red on right side „Nevi	Skin on right side blue on crying
Fortescue-Brickdale	M	9 mo.	Right	Normal		
Fischer, H.	F	9	Right	.....	Venous ectasia	Right side more fatigable
Fowler	F	10 mo.	Right	.....	Nevi	
Gesell	M	13	Right	Subnormal	Telangiectatic patches	Reversed asymmetry of epiglottis
Gordinier	M	65	Left	Normal	Nevi	Necropsy: left cerebral hemisphere +
Hornstein	F	2 mo.	Right	.....	Nevi	Necropsy: connective tissue hyperplasia
Hutchinson	M	5	Right	.....	.....	Necropsy: suprarenal, testicle, tonsil, etc.
Jacobi	..	3 mo.	Right			
Langlet	..	Adult	Right			
Logan	..	4	Right			
Masmejean	..	1	Right			
McGregor	M	10	Right	.....	.....	Necropsy: right optic thalamus +
Milne	F	19 mo.	Right	.....	.....	6 teeth on right side; 1 on left side
Milne	F	12	Right	.....	Telangiectasis	Right forefinger and thumb normal
Monod	M	19	Right	.....	Telangiectatic varicosities	Temperature + on right side
Möbius	M	14	Left	.....	Telangiectatic marbling	Pains in left arm
Mummery	M	5	Left	Normal	.....	Left side apparently grew faster in 2 years
Ollier	F	27	Right	Subnormal	.....	Temperature + on right side; cephalalgia
Pollosson	M	Adult	Right	.....	.....	Elephantiasis (?)
Redard	M	2	Right	Normal	Nevi	Hypertrophy right foot very marked; temperature right side +
Schiller	M	11	Left			
Telford	F	7	Right	.....	Nevi, varicosities	Rickets equal on both sides
Thomson	F	18 mo.	Right	Subnormal	.....	Thyroid absent? cretinism?
Tilanus	F	10	Right	Normal	Telangiectatic marbling	Right side stronger
Turbil	M	2 wk.	Left			
Wagner	M	11	Left	.....	Telangiectasis; blue pigment	

was reported normal in five cases and abnormal in three, the subnormality in two instances being associated with marked cranial asymmetry.

Of peculiar interest is the relatively frequent association of mental defect and hemihypertrophy. In 13 per cent. of the cases in the series this association is present. If the mental status could have been

TABLE 4.—CASES OF PARTIAL HYPERTROPHY

Author	Sex	Age	Side	Mentality	Skin Complications	Special Features
Adams	M	19	Right	.....	Nevus	Leg on right side 1½ inch longer
Adler	..	..	Right	.....	.....	Foot
Ballance	M	17	Left	Subnormal	.....	Head and certain toes of each foot
Bankhart	M	6	Right, left	.....	.....	Right side of face, tongue leg +; left arm +
Beck	M	28	Right	.....	.....	Thumb, forefinger, arm, right tactility —
Chassaignac	M	18	Right	.....	Varices	Right arm, leg
Chassaignac	F	..	Left	.....	Nevus	Leg only
Cohem	M	56	Right	.....	.....	Right leg; sugar tolerance +
Curling	F	..	Right, left	.....	.....	Right middle, ring finger 5½ inches; left thumb +
Davidson	F	1 mo.	Right	.....	.....	Leg
Foucher	M	..	Right, left	.....	Venous hyperemia	Left thorax, abdomen +; right fingers +
Friedberg	F	10	Right, left	.....	Telangiectasis, pemphigus, elephantiasis	Right leg adult size, right foot ++
Friedreich	F	16	Right	.....	Pigmentation; abnormal hair	Head: saliva + on right side, taste + on left side
Goldscheider	M	19	Left	.....	.....	Thumb, forefinger; sensation equal
Gruber	M	20	Left	.....	.....	Enormous hypertrophy of forefinger (16.5 cm.)
Hutchison	..	4 mo.	Left	.....	.....	Necropsy: left thymus +; left kidney 56 G., right 28 G.
Jacobson	F	3	Right, left	.....	Telangiectasis; abnormal hair	Right cheek, arm, hand +; left breast, abdomen, leg +
Lewin	M	26	Right, left	.....	Nevi; varicosities	Left side of face, arm, temperature, sweating +; right leg +
Osler	F	9	Right	.....	.....	Right arm adult size
Maschke	F	5	Left	Normal	.....	Synostosis of proximal phalanges
McConnell	..	7 mo.	Left	Normal		
Passauer	M	11	Left	Normal	.....	Marked facial asymmetry left cheek, tongue, teeth +
Pershing	F	19	Right	.....	.....	Trophedema ?
Prince	F	4 mo.	Right	.....	.....	Forearm, forefinger, thumb ++
Redard	M	3	Right	.....	.....	Temperature 2 to 4 degrees higher on right side
Reissmann	M	7 mo.	Right	.....	.....	Left cranium +; left dentition retarded
Speese	F	10	Right	.....	.....	Toe enormous size
Steffen	..	11 wk.	Right	.....	.....	Necropsy: hyperplasia; right hemisphere +
Widenmann	M	10	Right	Subnormal	Abnormal pigmentation	Right side of cranium +
Wittelshöfer	..	..	Right	.....	.....	Second, third toes +

determined in every instance, it is possible that the proportion might have been markedly increased. The facts clearly indicate that hemihypertrophy should be added to the list of developmental anomalies which bear some lawful relation to the incidence of mental deficiency. What is the nature of that relation?

Fig. 3.—Posterior view of subject in Figure 1, aged 13.

THE ETIOLOGY OF HEMIHYPERTROPHY

Since we do not know the true cause of hemihypertrophy, there is no dearth of etiologic theories. These "explanations" range from maternal impressions to internal secretions. A German mother ascribed the hemihypertrophy in her son to the fact that during pregnancy she had seen a youth of gigantic proportions in the market place. Presumably she saw only one side of this giant.

One writer attributes hemihypertrophy to an inherent tendency of the tissues to appropriate an excess of nutriment, but, as Ballantyne remarks, this statement is more ingenuous than ingenious.

Fortescue-Brickdale advances a venturesome biochemical theory. He suggests that the condition is due to an irregular distribution in the tissues of the anchoring substance which interacts with the glandular secretions which determine growth.

Another group of theories ascribes the hypertrophy to vascular lesions, calling attention to the frequency of vascular and skin complications and to the analogy of "naevus neuroticus unius lateris." One view is that an embryonic disturbance establishes a preeminence in the blood supply of one side or one part. Once established, this preeminence causes a permanent overgrowth of the favored side. This explanation is similar to that which ascribes right-handedness to the richer blood supply of the left hemisphere. Partial vasomotor paralysis has been suggested (Virchow); also incomplete development of the middle arterial coat. Pollosson regards a disturbance of the lymphatic system as the etiologic factor. But if the lesion is a vascular one, what is its origin? Is it hereditary?

There is no evidence that hemihypertrophy is hereditary. Bruns found only one case in the literature in which macrodactylism was present in several members of a family. There is one record of a family with apparently hereditary enlargement of the left hand, but we know of no instance in which two members of the same family or related members presented hemihypertrophy. To be hereditary the differential cause of a character must be present in the germ cell. Hemihypertrophy is congenital, but in origin it is probably embryonal and not germinal.

Hemihypertrophy may easily date back to some disturbance during the early embryonic period. The results of experimental teratology have shown that gravity, mechanical and other influences may induce monstrosities of various kinds. It is conceivable that a slight deviation from the harmony of physical influences surrounding the embryo may be competent to initiate a partial or unilateral hypertrophy in ways similar to those demonstrated in the laboratory. If the influence is teratologic, it may well go back as early as the fourth week in the embryonic period during the cleavage of the mesoblastic somites.

Wieland therefore regards hemihypertrophy as an embryonal malformation: "By continuous or intermittent proliferation processes in these somites during the intra-uterine life, the more severe types of congenital hypertrophy arise."

Leblanc similarly traces the lesion to the primitive embryonic neurotomes and their associated vasomotor center.

Greig takes the same general view and makes it more explicit. He argues that in the embryo all motor nerves may be regarded as trophic, because if they are destroyed or injured the parts supplied by them are more or less abnormally developed. Therefore it is to a central lesion or irritation of certain nerves brought about by intra-uterine

Fig. 4.—Posterior view of right leg showing areas of cutaneous congestion; subject aged 20.

meningitis or cerebritis that one must look for the origin of unilateral hypertrophy. Pathologic evidence adduced to support this interpretation is the enlargement of the foramina of certain nerves suggesting a previous inflammatory process.

If it is impossible to establish the existence of such definite extrinsic causes operating in the embryonic period, it will be necessary to search so far as we can among the deeper determinations of growth and development.

Growth depends on two groups of factors—internal and external. In the latter group are included the activators, hormones, vitamines,

food accessories and secretions. The action of these agencies, however, is general and mediated through the general blood supply. It is difficult to conceive how that action could be diminished or increased in a restricted manner in one member like the middle finger, or one side of the whole body to the exclusion of the other. Among the internal factors of growth are the so-called determiners which are already laid down in the fertilized ovum. It may be that for all paired or bipartite organs, instead of one determiner, there are two, or two groups of determiners, and the basis of asymmetry depends in some unknown way on an inequality of these primary determiners.

One investigator considered that in his case of hemihypertrophy the enlarged side looked fully a year older than the other, and that the ossification at the epiphyses of the forearm was, on that side, more advanced. Ossification is a criterion of physiologic age. A man is as old as his arteries, and a child, if we follow Dr. Rotch, is as old as the bones of his carpus. Does this mean that due to the difference in growth factors our boy with hemihypertrophy has three ages: his chronologic age, a right physiologic age and a left physiologic age? The fact that detention on the hypertrophied side is often precocious to the extent that there will be eight teeth on that side and none on the other is not altogether inconsistent with this fancy.

TWINNING AND ASYMMETRY

It is natural that a discussion of the etiology of hemihypertrophy should finally bring us to problems of double physical personality and twins. Further researches into the biology of twinning may bring the remarkable phenomenon of unilateral hypertrophy more completely within our comprehension; may even prove it to be on closer scrutiny more frequent and less anomalous than we had supposed. Indeed, even now, all things considered, the real marvel is not the occurrence of hypertrophy but the fact that hemihypertrophy is such an extreme rarity.

"By twinning we mean production of equivalent structures by division." This statement is taken from the biologist Bateson, who regards the power to divide as a fundamental attribute of life. The tendency to symmetry, to bilateral equivalence or mirror imaging is so general that it also must be regarded as a fundamental of biologic mechanics. Hemihypertrophy accordingly may be conceived as some profound inaccuracy in the natural process of developmental duplicity. It is not as monstrous as the double monster, but it may have a related morphogenesis. At any rate, we can safely assume that hemihyper-

trophy is not an artifact really consisting in a hemi-atrophy. It is evidently a mild unilateral gigantism of an individual whose lesser somatic half is normal.

In a certain biologic sense we may regard every bilateral individual as being a pair of twins. H. H. Newman, in his fascinating work on the "Biology of Twins," holds that monozygotic twinning—where a single egg produces two offsprings—is "a phenomenon that should be considered as only a phase of the much more general phenomenon of symmetrical division. The development of the right and left hand homologous organs in a bilateral organism is

Fig. 5.—Cast of palate and upper teeth.

essentially a twinning process." This author also observes that "the whole matter of bilateral development appears to be quantitative in nature, in that the same type of process may go not so far or further than normal." Just as there may be an inhibition of the normal culmination of the process of bilateral division (as in the single median cyclopic age), so there is frequently an excess of division resulting in the two bilateral structures becoming completely segregated, as when a single individual develops two heads or two tails, while the remainder of the organism is a more or less normal individual. Newman, like Bateson, regards the phenomenon of twinning as a fundamental process which is almost universal in the field of biology.

From this point of view, hemihypertrophy may be interpreted as an atypical or imperfect form of twinning, a variant of the same process which may produce a double-headed monster or a perfectly, ordinary normal individual—an ordinary individual being an organism in whom there has been a precisely baianced inhibition on the biologic process of bilateral doubling.

The actual mechanism of this regulative control over symmetry of size and form remains somewhat enigmatic in spite of much biologic investigation. Davenport regards size or stature as a unit character of inheritance subject to Mendelian principles; but this does not assist us in interpreting the curious stature anomaly embodied in hemihypertrophy.

Newman has made suggestive researches into heredity and organic symmetry in armadillo quadruplets. He has noted some cases in which "one lateral half of the body has quite a different number of scutes from the other half, and one of these halves resembles the maternal condition." Since each set of quadruplets have the same genetic constitution as they arise from one zygote, he concludes that some irregularity in the mechanism of the mitotic cell-division is responsible for the anomalies of symmetry. This factor is by no means a simple one. "Now in the armadillo there are many evidences of a system of symmetry common to all of the quadruplets, upon which has been superimposed a secondary symmetry system between twins. This in twins is more or less obliterated by a tertiary symmetry between the antemeric halves of the single individual."

R. G. Harrison discusses rules of symmetry in his monograph "On Relations of Symmetry in Transplanted Limbs." This study is based on 462 cases of grafting of limb buds in amblystoma punctatum. He agrees with Morgan that the potential factors of symmetry reside in the constitution of the egg. "It is the intimate protoplasmic structure that underlies symmetry." Likewise reversal of symmetry. "As an alternative to the hypothesis of rotation, we might consider reversal as due to reversal of molecular asymmetry according to analogy with the behavior of optically active compounds." "There is an analogy between the production of enantiomorphic limbs and the production of situs inversus viscerum, as effected by Speeman. Either the reversal may be due to reversal of the intimate structure, or it may take place in spite of the intimate structure through the direct action of mechanical factors on the individual parts of the differentiating system." [1]

1. Speeman obtained a large number of twins in Triton by constricting the eggs in segmentation stages or in early blastula. In many of the cases one individual, usually the right, developed complete situs inversus viscerum.

Another form of asymmetry, even more startling and drastic than hemihypertrophy is that of gynandromorphism. A gynandromorph is an animal that is male on one side and female on the other. This differentiation may include the reproductive organs, gonads and ducts. Usually it is longitudinally bilateral, but it may be anteroposterior.

Fig. 6.—Roentgenogram of hands of subject at age of 13.

This curious phenomenon is most frequent in insects but has been reported in birds and in a few mammals. A beautiful case was described in a mutillid wasp in which the male half of the body was black and winged like the male while the female half was a rich red and wingless.

The problem of gynandromorphism has been extensively studied by F. H. Morgan and C. B. Bridges and reported in their contributions to the genetics of *Drosophila melangaster*. They found one gynandromorph among every 2,200 flies. The authors consider a gynandromorph a hybrid whose genes are carried by the sex chromosome; and they give definite evidence that the peculiar sex mosaic condition is due to an elimination of one X chromosome, usually at some early division of the segmenting nuclei.

MENTAL DEFECT IN RELATION TO HEMIHYPERTROPHY

The foregoing discussion indicates that hemihypertrophy, even though it is frequently associated with mental defect, should not be classified as a stigma of degeneracy. It is congenital, but it is apparently not due to an intrinsic imperfection in the genetic constitution of the germ cells. A stigma of degeneracy would denote such an imperfection. Hemihypertrophy rather denotes an abnormal deviation in morphogenesis and is to be classified as an anomaly of development.

Anomalies of development are found with a high degree of frequency among the feebleminded. Even in growth of stature they show significant departures from the normal. As Goddard found, "low grade idiots have not only a disturbed brain function but their entire organism is disarranged and growth processes upset." In the imbecile the same is true but to a less extent. While mild asymmetry in structure is proverbially normal, pronounced asymmetry is certainly more characteristic of the mentally defective as a class. In a careful study, La Page measured the skull conformation in a series of 198 cases of feeblemindedness and found lateral asymmetry in 158, or 80 per cent., and, what is very interesting in this connection, 122, or 80 per cent. of these cases, showed the hypertrophy to be on the right side. This is in harmony with the ratio between right and left in our series of forty cases of total unilateral hypertrophy. Is it not probable that there is some fundamental similarity or even identity in the causes which produce pronounced cranial asymmetry and hemihypertrophy?

Little is known about the neuropathologic facts. In those cases which have come to necropsy the histologic study of the hypertrophied organs shows that the increase in bulk is due chiefly to an overgrowth of connective tissue. This fact again establishes a possible connection with feeblemindedness. Indeed there is a clinical form of mental defect which is known as hypertrophic amentia, which consists in an overgrowth of interstitial tissue. It may produce a skull, square in shape, 25 inches in circumference. Although this type of amentia is rare, sclerosis (or overgrowth of neuroglia) occurs frequently. Dr. Wilmarth

examined the brains of 100 feeblminded children and found evidence of sclerosis in fully one fourth of the cases. Tredgold believes that in some of the cases it is probably due to vascular lesions.

Since hemihypertrophy consists in large measure of a diffuse sclerosis, it may be that we have the production of mental defect in those cases where the sclerosis involves one or both of the hemispheres. One cause would then be adequate to explain both conditions when they are associated.

Fig. 7.—Roentgenogram of feet of subject at age of 13.

Furthermore, if the exciting cause of these vascular lesions in the great majority of cases, as Greig suggests, is an intra-uterine meningitis or cerebritis, we have an additional factor for the production of mental defect.

Dr. Gordinier's case of unilateral hypertrophy came to necropsy, but the brain, unfortunately, did not come to microscopic section. The viscera in general showed a diffuse interstitial connective tissue hyperplasia. The left cerebral hemisphere on inspection was distinctly larger than the right, and on measurement proved to be 1 cm. greater in its anteroposterior and 1.25 cm. greater in its transverse diameter. The intelligence of the subject was reported normal.

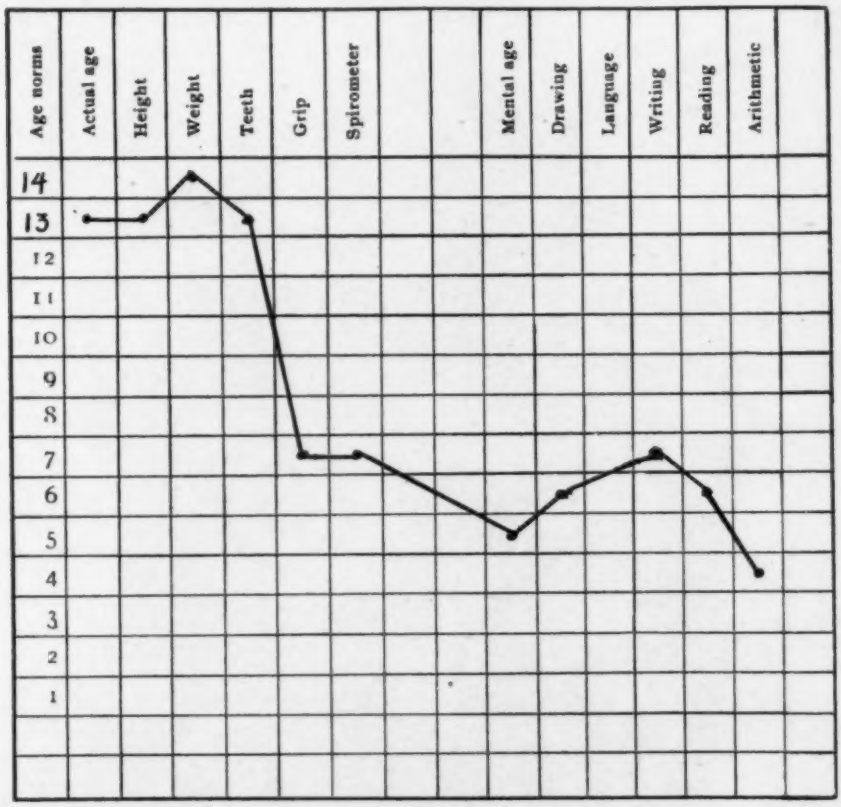

Fig. 8.—"Norm graph" showing standards of physical and mental development.

The production of actual mental defect in any particular case may therefore be contingent on the degree to which neurogliosis has taken place, and to the degree of compensatory development in unaffected nerve tissue.

It is possible that there are different causes for different cases of hemihypertrophy, and it may be that each case represents its own distinctive interaction between some intimate defect in the symmetry determiners and the environment of the multiplying cells. It is tempting, however, to relate true hemihypertrophy and certain not infrequent

forms of cranial asymmetry to some quantitative abnormality in the chromosomal mechanism, which produces not only a deviation in the process of bilateral twinning, but also may upset normal tissue relations, increasing the proliferation of certain cells at the expense of others.

This is not out of harmony with Boveri's suggestion that an imperfect or irregular division of the chromosomal complex may, through loss of specific chromosomes, remove regulative inhibitions which permit certain inimical or normally suppressed cells to run wild.

The frequent association of hemihypertrophy and of cranial asymmetry with mental defect and the consistent preference in both conditions for enlargement of the right side suggests some lawful grouping of causative factors. It is quite possible that the origin of certain altogether obscure cases of secondary amentia may lie in an undetectable but decisive imbalance of the fundamental process of twinning which follows fertilization.

Whatever our final hypothesis may be, we seem to be warranted in the broad conclusion that true hemihypertrophy consists in an erroneous deviation from the normal course of development in the embryo, and carries with it, as so many anomalies of development do, the possible penalty of mental defect.

SUMMARY

Hemihypertrophy is a unilateral enlargement of one side of the body, and is one of the rarest among developmental anomalies.

A case associated with mental deficiency is described. Physical and mental measurements were made when the subject was 13 years old and repeated at the age of 20.

A comprehensive tabulation of cases to date (Table 3) shows a total of forty cases recorded in medical literature, of which nineteen were male, twenty-seven on the right side, nineteen with skin complications and five with mental defect.

A less complete tabulation of partial and crossed hypertrophies, including thirty cases, is presented for comparison in Table 4.

In a discussion of the etiologic theories for hemihypertrophy, preference is given to the view that hemihypertrophy is not a hereditary character but a morphogenetic anomaly dating back to an early embryonic stage.

Hemihypertrophy is interpreted as a form of asymmetry due to a possible deviation in the normal process of twinning.

The complication of mental defect is attributed to an abnormality in the process of bilateral twinning which involves a disturbance of normal tissue development.

Possible relations of certain cases of mental defect to cranial asymmetry and intra-uterine meningitis are suggested.

The value of further biometric and clinical studies in asymmetry is indicated.

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EYE SIGNS IN INTRACRANIAL TUMORS OF THE ANTERIOR FOSSA

WITH REPORT OF TWO CASES*

ALBERT B. SIEWERS, M.D.

SYRACUSE

Retrobulbar neuritis with central scotoma and primary optic atrophy on the side of the lesion and concomitant papilledema in the opposite eye as a syndrome in intracranial tumors of the anterior fossa was stressed by Kennedy [1] in 1911. In the same year Paton and Holmes, in a study of brain tumors, state:

> We have examined a case in which a basal tumor compressed one optic nerve before its exit through the optic foramen and produced a retrograde atrophy of the nerve fibers and ganglion cells, and sclerosis of the nerve and disc. There was no edema . . . In the other eye there was typical papilledema.

Similar cases had been observed in isolated instances. Willbrand and Saenger [2] quote Seydel, who observed in a man aged 47, slight paresis of the right half of the body and trembling of the extremities, beginning in the right arm. At the age of 32, he had received a blow on the head followed by unconsciousness for several weeks. Ophthalmoscopic examination revealed papilledema in the right eye and in the left a pale nerve head. There was an oval shaped absolute central scotoma on the left. Removal of an extradural fibroma in the anterior fossa brought about regression of symptoms.

The same authors cite two other cases—one of frontal tumor on the under surface, more on the left than on the right. The left optic nerve was directly compressed. The result was primary atrophy of the left and intense neuritis of the right optic nerve. The other was a case of right frontal tumor. The right papilla appeared normal, but the patient was blind in this eye. The left disk showed a swelling of 2 diopters. Visual acuity was good on the left.

REPORT OF CASES

CASE 1.—*History.*—About Sept. 1, 1920, C. J., a fire department chauffeur, 38 years old, began to complain of pain in the right occipital region; it recurred

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*From the psychiatric and Surgical Service of the Johns Hopkins Hospital.

1. Kennedy, Foster: Am. J. Med. Sc. **142**:355 (Sept.) 1911; J. A. M. A. **67**: 1361 (Nov. 4) 1916.

2. Willbrand and Saenger: Centralbl. Chir. 1896, No. 3.

spasmodically. Soon after, he began to have attacks of uncontrollable coarse shaking of the right arm. He was several times incontinent of urine while going about his business and incontinent of feces once while in bed. He was also impotent. By the middle of October he had to give up work.

After December 1, he vomited several times without nausea and with considerable force. During the week preceding his admission he had difficulty in walking, and seemed rather dull. On December 20, he came to the Henry Phipps Psychiatric Clinic with this dulness, occipital headache, and attacks of trembling of the right arm. He was admitted December 23.

He was correctly oriented and showed slight "immediate memory" defect; that is, his retention was only four digits, probably accounted for by poor attention and poor cooperation. At times he was confused and restless; he would get in and out of bed repeatedly. He said: "I feel fine except when I have that pain."

Fig. 1.—(Case 1).—Tumor on under surface of frontal lobe.

Examination.—The patient lay in bed with eyes and head turned toward the right. He tended constantly to roll out of bed on the right side. He could smell camphor and peppermint, but could not name them or tell their nature. (The left eye had been blind for some time and had been more prominent than the right since the beginning of the illness.) The left disk was atrophied. There was beginning papilledema in the right eye. The pupils were regular, the right slightly smaller. The right reacted well to light and accommodation, but the left did not react. There was a coarse, rhythmic tremor of the right arm; rate, four to five per second. The finger-nose test showed some ataxia and there was dysdiadochokinesis of both hands. Abdominal wall reflexes were absent. The cremasteric was active on both sides. The deep reflexes were normal and the plantar response flexor on both sides. The gait was reeling, the patient falling backward and to the right. He could not stand unsupported.

During the examination the patient appeared to have several petit mal attacks, of which he had no realization. No disorder of the internal organs was found.

The Wassermann reaction on the blood and spinal fluid was negative. The fluid showed increased globulin and 3 cells. The roentgen-ray picture of the skull was negative.

Because of the headache and vomiting, repeated incontinence and dulness, protrusion of the left eye, atrophy of the left and edema of the right nerve-head, tendency to deviation to the right, the tremor attacks of the right arm and postural difficulty, a diagnosis of left frontal tumor was made, and the patient was transferred to the department of surgery.

Fig. 2.—(Case 2).—Tumor, arising from dura, located under right frontal lobe.

Treatment and Course.—On December 29, Dr. W. E. Dandy found that only the right lateral ventricle filled with air. Two days later he exposed the left frontal lobe and found a diffuse dural endothelioma with a large mass of tumor on the under surface of the frontal lobe. The tumor could not be entirely removed. Figure 1 shows the size of the removed part.

The patient made an uneventful operative recovery and after two weeks was able to go home. Later he received several radium treatments. On June 1, 1921, he reported feeling perfectly well and was back at work in the fire department, but not driving a car.

Laboratory Findings.—The dural surface has the same appearance as that part of which spread diffusely over the under surface of the dura. There was a "crust or frosting" of tumor half an inch thick, adherent to the dura. It extended to the longitudinal sinus above, as far back over the occipital lobe as one could see, down for a short distance over the temporal lobe and nearly to the midline in front.

Case 2.—*History.*—W. S., aged 51, a laborer, formerly an electrician, was admitted to the Johns Hopkins Hospital, on Aug. 6, 1920, complaining of "spells"

accompanied by an odor of sewer gas. Eight years ago he received an electric shock from an 18,000 volt circuit. Some "black blood" discharged from his nose a week later. He continued at work but "nervousness" not complained of previously now caused changes in occupation more or less frequently.

In May, 1918, he suddenly became "light-headed" and noticed a strong smell of sewer gas. He was working on a pipe line at the time and paid no attention to the occurrence. Attacks similar to this then came on two or three times daily and lasted two or three minutes. In the attacks there were color hallucinations. Any object that he happened to be looking at seemed to become red or green as if looked at with a colored glass. These hallucinations gradually disappeared.

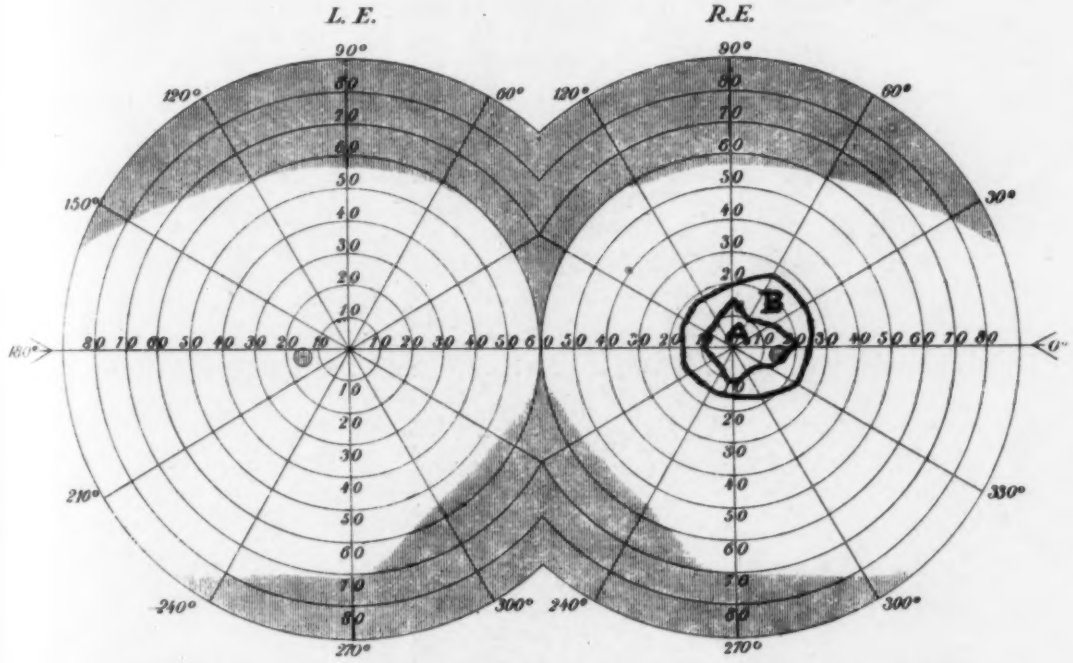

Fig. 3.—(Case 2).—Fields of vision; *A*, absolute scotoma; *B*, a larger bordering scotoma for red and green.

The visual fields of C. J. (Case 1) were not charted. He was totally blind in the left eye. The right visual field was normal.

In February, 1919, he had an attack that started the same way but passed into a generalized convulsion with loss of consciousness. He had had five such attacks.

Since July, 1919, he had complained of headache and of inability to see with the right eye an object directly in front of him. He had become more irritable, had lost 35 pounds in weight, and for the last year had had no sexual desire or power. (It will be noted that the patient in Case 1 had also lost his sexual power.)

Examination.—The patient recognized test odors. The pupils were normal. Vision was good except for a central scotoma on the right. The right disk

was beginning to atrophy, especially on the temporal side. There was beginning papilledema on the left. The abdominal reflexes were present; the left was reduced and easily exhausted. The cremasteric reflexes were active and equal and the deep reflexes normal. The plantar response was flexor. There was dysdiadochokinesia of the right hand.

The internal organs were normal. The blood and spinal fluid Wassermann reactions were negative. The fluid showed an increase in globulin. Roentgen-ray examination revealed increased density over the right frontal region.

Treatment and Course.—A diagnosis of right frontal neoplasm was made, and on Aug. 11, 1920, Dr. W. E. Dandy exposed the right frontal lobe. A tumor arising from the dura under the right frontal lobe and extending backward was enucleated. It was very vascular and weighed 62 gm. There was terrific bleeding; the patient was shocked and died the following evening.

COMMENT

It will be noted in the two cases that the eye signs as described by Kennedy and others were very prominent and of considerable significance in the diagnosis of the condition and of the localization of the lesion.[3]

405 Fayette Park.

3. In addition to the references given the following may be of interest: Paton, Leslie: Brain **32**:64-91, 1909-1910; Paton and Holmes: Ibid. **33**:420, 1916-1917; Willbrand and Saenger: Die Neurologie des Auges, **2**:535 and 586.

RESULTS IN TWO HUNDRED AND FIFTY-ONE CASES FIVE YEARS AFTER ADMISSION TO A HOSPITAL FOR MENTAL DISEASES

EARL D. BOND, M.D.

PHILADELPHIA

A study has been made of what happened to two hundred and fifty-one consecutive women patients in the five years following their admission to the department for mental and nervous diseases of the Pennsylvania Hospital. We have records of isolated cases that have been followed for a life-time, but they lack the authority that comes from unselected consecutive cases. The fact that this as a private hospital has insisted on having responsible families and friends stand behind its patients has made possible this laborious undertaking.

The charts and tables show the condition of each of the 251 patients through five years. In the charts an individual can be followed by reading down a numbered column; psychosis unimproved is shown by close hatched lines, improved (usually fully able to look after herself at home) by lines more widely spaced, and recovery is shown by white spaces closed in at the bottom by a line indicating the time of the last report. More concisely, the more cross lines the more illness.

The list is continuous, but for convenience in reference has been broken into four parts—A, B, C, D.

RECOVERIES

In Group A Patient 1 is shown in the chart as sick for three and a half months, then as making a good recovery which she has consistently held for nearly five years. At the end of the fourth year after admission she was found by the follow-up nurse at her former home and appeared well. At the end of the fifth year when the nurse arrived she was out marketing, but her mother and the neighbors said that she was in very good health. She was a married woman, aged 30, who had had one child before admission and none since. Other cases, up to Patient 30, follow the same paradigm; the illness has varied within narrow limits, except in Patient 8. And yet, the obstinacy of the attack in this instance, over three years in a patient aged 53 with a hemiplegia, has given way to recovery which has restored a woman of fine character and high ability to a world which needed her. On leaving the hospital well she went to a distant state and saw her only son embark for France. She awoke one morning to find her husband lying beside her bed unconscious from the same kind of cerebral hemorrhage from

which she once suffered and nursed him through a long and hard illness to his death, after which she broke up her home and has acted as administratrix of the estate. Patient 30 herself, a woman of 60, made a good recovery and died at home over two years later of "cardiac asthma" without any mental symptoms. Patients 31, 32 and 33 had long drawn out cases of involutional melancholia with good endings; all reached the point of emaciation; Patient 33 remained at this hospital during the whole psychosis; Patient 31 recovered at the Norristown State Hospital; Patient 32 tried three other hospitals and recovered under the care of a nurse at home. She is "as well as ever in her life, but much more amiable." Patient 34 after two short attacks and some symptoms which showed after she went home has been very well for

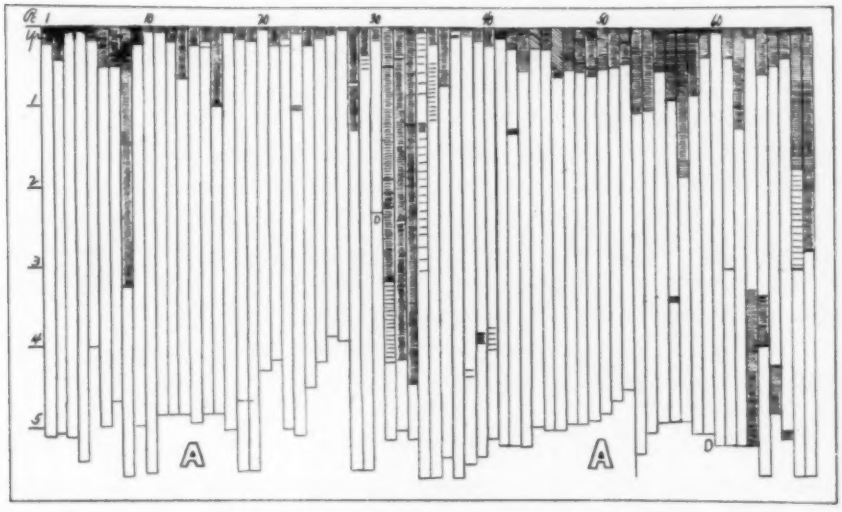

Chart 1.—Review of five year period.

over two years and is holding a much better position than her abilities before the first attack seemed to warrant.

The amazing and cheering thing in these first sixty-eight cases is the spectacle of normal life regained—even for a five-year period. The total is over two centuries and a half. Sixty patients have met the test of "sustained self-adjustment to life outside" and do not at present show signs of breaking down. The facts found about these sixty-eight out of 251 patients show well enough what "recovery" can mean and whether or not it is worth while.

Table 1 gives a list of the sixty-eight patients in the same order as on the chart, including the age, the diagnosis (as of present date) and features which are unusual. As these studies bear on the social

importance of a recovery, an effort has been made to find the number of children born since the last admission, and these are noted with the number born earlier.

A summary of diagnoses for this group of sixty-eight maintained recoveries shows that there were 40 manic-depressive psychoses cases, 14 unclassed, 6 involution melancholias, with an average hospital treatment of over two years, 3 psychoses with infections, 3 psychoneuroses, 1 psychosis with morphia and 1 dementia praecox,

These patients had had sixty-five children before the attack for which they were admitted, four of whom had died. Since the attack only two children had been born to them, according to our information,

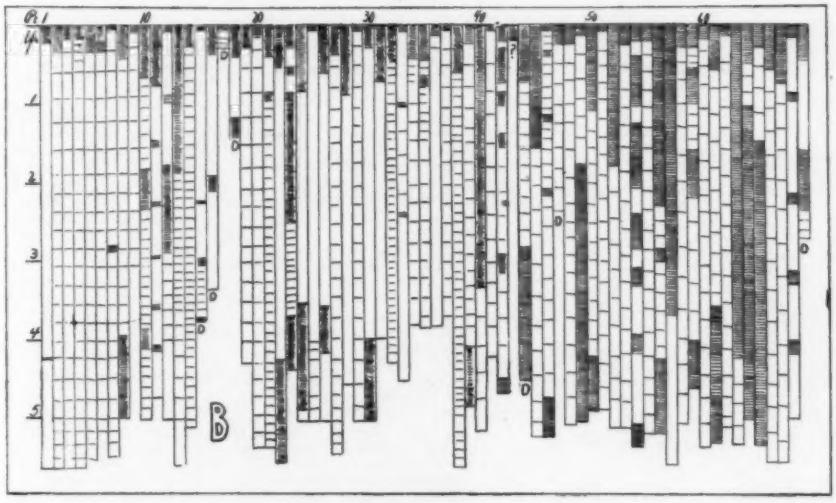

Chart 2.—Review of five year period.

which is quite accurate. Three patients, after waiting for four or five years after recovery, were married.

PARTIAL RECOVERIES

Cases collected under B show as striking a tendency to improve as do those under A, but are as strikingly differentiated by coming to a partial or poorly maintained adjustment. In the sixty-nine cases here grouped the improvement is manifested as early as in the sixty-eight of the first group, and all but five have become capable of self-support.

Patient 1 was sick three months and for five years since has been at home, capably managing a household of four people without help, but "nervous." Four years after recovery she had a severe cold accompanied by headache and delirium for two weeks.

TABLE 1.—AGE, DIAGNOSIS AND UNUSUAL FEATURES IN CASES OF SIXTY-EIGHT PATIENTS RECOVERED

No.	Age	Diagnosis	Comment
A1	30	Manic depressive	One child before, none since
A2	40	Manic depressive	One child and one induced miscarriage before, none since; back fractured by leaping out of window; good physical result
A3	62	Manic depressive	Single
A4	58	Manic depressive	Three children before
A5	26	Manic depressive	Married four years after admission
A6	40	Manic depressive	Two children before, none since
A7	25	Manic depressive	Single
A8	53	Manic depressive	Hemiplegia; one child before, none since (see text)
A9	26	Psychosis with infection	One child at beginning of this attack, none since
A10	35	Manic depressive	Three children before, 1 died, none since
A11	30	Manic depressive	Single
A12	25	Unclassed	Married at end of fifth year of recovery
A13	26	Manic depressive	No children
A14	55	Manic depressive	Four children before attack of whom three died; living child gives Wassermann +
A15	38	Unclassed	Divorced; no children
A16	35	Manic depressive (infection)	One child before attack
A17	46	Manic depressive	Single
A18	28	Unclassed	Divorced, no children
A19	29	Dementia praecox	Single; two years before admission her father died; months before admission her minister died; the patient showed unusual grief. For five months before admission and two months in the hospital she was reticent, uninterested in her surroundings; expressed somatic delusions (stoniness and iciness); thought that she made other people sick; speculated on telepathy, on how she could hold "the dream life." She had been fond of her father, but said she had no real affection for her mother; recovery after a little over two months in hospital; has been carefully followed since; she has entered and graduated from college and is holding a responsible position. She still thinks of her illness as a "fight between the real and the unreal"
A20	19	Psychosis with infection	Single
A21	50	Unclassed	One child before attack none since
A22	43	Involutional melancholia	No children
A23	50	Involutional melancholia	One child; menopause at 43
A24	43	Involutional melancholia	Single
A25	58	Manic depressive	No children
A26	24	Unclassed	One child before
A27	47	Manic depressive	Married, has had no children
A28	34	Manic depressive	Four children before attack, none since. This patient made first a recovery which lasted only a month, but after a relapse of six months has remained well four years and is now a competent housekeeper for her family of five, and carries on and enjoys her social duties
A29	19	Manic depressive	A deaf mute; has shown for five years all the ability and poise which she had before the attack. As the deafmutism can be considered separately from her mental disturbance it seemed fair to mark her "recovered." Patients with a mental upset on a psychopathic inferior basis have been called "improved" when the psychosis cleared up
A30	66	Unclassed	Four children; died of asthma long after recovery
A31	48	Involutional melancholia	A widow with two children
A32	43	Involutional melancholia	Married; three children before, none since; in recovering increased from weight of 70 to 125 pounds
A33	50	Involutional melancholia	Married, one child before, none since
A34	19	Psychoneurosis	Single; a recovery with a gain of efficiency and poise over previous condition
A35	17	Unclassed	She was married five years and nine months after admission
A36	44	Morphia hallucinosis	Single; her brother reports that she has kept house without difficulty and has not to anyone's knowledge returned to her drug
A37	20	Unclassed	Married a year after recovery; deserted by her husband after the birth of one child; she is working as a saleswoman and supporting her child, but she is "still rather a rattlebrain"
A38	50	Manic depressive	Single; a depression which apparently replaced convulsions, and was filled with desperate suicidal attempts. She has been well, fat, and has held a good job for five years; in the summer of her fourth year she was tired and weak
A39	17	Manic depressive	Single; two sharp attacks of excitement with good recovery

TABLE 1.—AGE, DIAGNOSIS AND UNUSUAL FEATURES IN CASES OF SIXTY-EIGHT PATIENTS—(*Continued*)

No.	Age	Diagnosis	Comment
A40	19	Manic depressive	Single; very well since early recovery with the exception of a mild melancholia in the fourth year; always well employed
A41	19	Psychoneurosis	Single; hysteria
A42	51	Manic depressive	Single
A43	12	Unclassed	Hallucinated or suicidal; now doing well in high school
A44	23	Unclassed	One child * (p. 393)
A45	54	Manic depressive	Widow; one son
A46	51	Manic depressive	Widow; no children
A47	21	Unclassed	Single * (p. 394)
A48	47	Manic depressive	Two children before
A49	34	Manic depressive	One child
A50	26	Manic depressive	One child; after the chart was drawn, the patient had another attack which is not indicated
A51	44	Manic depressive infection	Single
A52	58	Manic depressive	Seven children. She has had another attack since the chart was made; for several years she has been very deaf and sensitive; otherwise she has all her life seemed to be a hard working, well balanced individual
A53	43	Manic depressive	Seven children
A54	31	Manic depressive	One child
A55	23	Manic depressive	Single
A56	22	Manic depressive	Married; no children
A57	24	Manic depressive	Married; one child
A58	27	Manic depressive	Single; in better health and earning much more than before the attack
A59	45	Manic depressive	Single; died five years after recovery, of pneumonia, without any recurrence of mental symptoms
A60	35	Unclassed	Married; no children
A61	23	Manic depressive	One child; this patient was followed three years, during which time she actively assisted her husband in his store at Atlantic City; they moved suddenly and we have not been able to locate them
A62	34	Unclassed	Sent from here and from a state hospital unimproved; recovered when she heard that her husband was unfaithful* (p. 393); two children before admission, none since
A63	45	Manic depressive	Three children. The new attack was associated with virulent mouth infections
A64	39	Unclassed	Single
A65	21	Manic depressive	Single
A66	47	Manic depressive	Single
A67	24	Psychosis with infections	One child
A68	30	Psychosis with infections	One child before admission, one child since

* Bond, Earl D.: A Review of the Five-Year Period Following Admission in One Hundred and Eleven Mental Patients, Am. J. Insan. **76**: 385-394 (Jan.) 1921.

The cases of patients 2 to 8 illustrate the clearing from psychoses with residual symptoms, which either were produced by the psychosis or were part of an evident congenital or earlier acquired inferiority. Patient 2, for instance, backward, with many physical signs of an old anterior poliomyelitis and irritable, at the age of 25 became depressed and deluded; she recovered from this acute mental attack in 4 months and has been at home 5 years since in a cheerful, clear, but always partially dependent condition. Patient 3, sexually delinquent, at 18 had a hallucinosis, from which she recovered in two months; for five years since she has been self-supporting, and, according to the society which has followed up her case, she has been doing extremely well. Patients 3 and 4 made prompt recoveries from acute psychoses, but were left with a lack of insight (3) and eccentricities (4), which prevent our regarding them as well rounded recoveries. Patient 7 is probably

the most efficient worker of the 251 patients, and her efficiency has increased since her mental attacks; she visits the hospital regularly and is nervous and restless. Patients 10, 15, 33, 42 and 44 show alternating phases of manic-depressive psychosis; all of them could be called recovered at many given points, and could swell a recovery rate. Patients 13 and 40 are recovering after long illnesses. Patient 25, an able business woman, has kept in close touch with the hospital since her recovery from a severe delirium at childbirth; while she has all her former ability, she has in the last year had attacks of petit mal.

This group of seventy-one patients (B) as a whole seems efficient but queer, while Group A seems well balanced. No new therapeutic procedure could be expected to attain much better results with Group A, but in Group B is much valuable material to be salvaged. Those patients who have gone quickly from one phase of manic-depressive psychosis to another have made the most unsatisfactory social record because of disturbances at each commitment and because two have made unwise marriages and each has given birth to a child.

This group of sixty-nine patients who showed a tendency to improve to the condition of self-support gives diagnoses of manic-depressive cases in 27 instances, involutional melancholia in 5 and dementia praecox in 9 cases, 15 remaining unclassed; 8 psychoses in psychopathic inferiors, 2 psychoneuroses, 2 psychoses with somatic disease and 1 alcoholic psychosis.

These patients had seventy-three children before they were admitted, of whom eight had died, one by suicide, and two have become state charges (insanity and desertion). Since admission they have had seven children, though by their very inclusion in this group the mothers are in no condition to look after them.

UNIMPROVED UNTIL DEATH

In Group C are included the fifty-one patients that showed no improvement and died, twenty-one of the deaths occurring away from this hospital. This group is not charted; 2 deaths occurred at over 90 years of age, 16 deaths between 90 and 60, 22 between 60 and 40 and 10 below 40.

Of the thirty deaths in hospital, nineteen resulted from recognized and chronic progressive diseases. Six others were covered by necropsy; three of these were described in a previous paper * and two in Dr. Strecker's † paper on "Late Catatonia." The other showed marked degeneration of the liver, kidney and cord. Three other patients died within eight days of infections existing before admission. The two remaining deaths were due to exophthalmic goiter and lobar pneumonia, but were favored by the exhaustion due to psychoses.

TABLE 2.—FINDINGS IN GROUP B IMPROVED

No.	Age	Diagnosis	Comment
B1	38	Unclassed	Three children before, none since
B2	25	Psychosis in inferior	No children
B3	18	Psychosis in inferior	Sex delinquent; no children
B4	26	Dementia praecox	No children
B5	57	Manic depressive	One child before (suicide); none since
B6	44	Manic depressive	No children
B7	34	Unclassed	Three children before; none since
B8	48	Dementia praecox	No children
B9	31	Psychosis in inferior	Married since; (?) children
B10	37	Manic depressive	No children
B11	49	Manic depressive	Two children; none since
B12	38	Manic depressive	Four children before, 1 child since
B13	42	Involutional melancholia	No children
B14	34	Manic depressive	One child before, none since
B15	51	Manic depressive	Eleventh attack; death in fourteenth attack; single
B16	27	Manic depressive	Fever (105 F.) with congenital aortic stenosis; recovery from this attack and another two years later; death at home after three years of pneumonia. Married; no children
B17	42	Involutional melancholia	Some improvement before death of uterine cancer; widow; one son in state hospital; dementia praecox
B18	19	Unclassed	Hyperthyroidism; death in second attack in state hospital; single
B19	45	Dementia praecox	Improved enough to go home where she did poorly; under osteopathy for three months, then fairly well, but very peculiar; three children before, (?) since
B20	16	Psychopathically inferior	Sex offender, sterile; deaf; has been self-supporting under constant supervision
B21	40	Manic depressive	Twelfth attack; seven children before
B22	30	Manic depressive	Second attack; one child born before her admission here; now under care of Children's Aid; husband alcoholic
B23	28	Manic depressive	Married; no pregnancies
B24	41	Manic depressive	Married; four children
B25	36	Psychosis with somatic disease	One child, none since; described in text
B26	25	Unclassed	Single
B27	57	Alcoholic psychosis	Single
B28	30	Unclassed	Eccentric, but causes no trouble at home
B29	32	Unclassed	Single on admission but three years later married and had one child, husband died shortly after; patient is nervous but well; supports herself
B30	29	Manic depressive	Second attack; first attack was postpuerperal; has had three children; since admission one has died and another has been born
B31	57	Involutional melancholia	Single
B32	48	Involutional melancholia	Married; eight children, four of whom are dead; menopause at 46
B33	18	Manic depressive	Third attack; married three years after admission and has a baby
B34	21	Unclassed	Single; well employed
B35	23	Dementia praecox	Married; one child
B36	33	Psychosis with constitutional inferiority	Fourth attack; had two children (one illegitimate) before admission; divorced
B37	50	Paranoid condition	Single; queer and nervous but a good worker; more than self supporting
B38	42	Unclassed	Married; two children before this attack, none since. While she has done the housekeeping for herself and children, she has constantly quarreled with her husband and twice sent him to jail
B39	23	Dementia praecox	An attack with a pseudorecovery; at home seclusive and without insight until an attack four years later, for which she is still under treatment; single
B40	38	Involutional melancholia	Single; a three-year attack with "recovery" marked by lack of insight, but ability to do her work at home
B41	52	Psychoneurosis	A widow with two children; while much better when she left the hospital and able to gain ten pounds, she is now a dispensary rounder
B42	26	Manic depressive	The circular type, barely getting an emotional balance for a week or so between attacks; in her excitements she has been much in the newspapers, has made an unfortunate marriage, and has had a baby
B43	20	Dementia praecox	Lost after discharge from another hospital; single
B44	29	Manic depressive	Married; no children; died in second attack
B45	40	Manic depressive	Single
B46	18	Dementia praecox	Single

TABLE 2.—FINDINGS IN GROUP B—(*Continued*)

No.	Age	Diagnosis	Comment
B47	67	Unclassed	Married; no children; died of heart failure after two years of good health at home
B48	41	Constitutional psychopath...	Married; no children
B49	55	Unclassed	Single
B50	35	Manic depressive	Single; able to work as a private nurse for three years
B51	55	Manic depressive	Fifth attack; eleven daughters of whom one died; at home doing much good work for her church
B52	41	Psychosis with somatic disease	After birth of second child;† recovered after seventeen years' illness, went home and came back for a second admission; this time she gradually completed making adjustments to new conditions and was able to leave the hospital and remain outside
B53	58	Manic depressive	Single
B54	63	Manic depressive	Manic attacks; one child
B55	18	Constitutional psychopath...	Single; no children
B56	45	Manic depressive	Circular; single
B57	20	Manic depressive	Long attacks; for the last fifteen months a pupil nurse in a New York hospital
B58	54	Manic depressive	Depression; three children
B59	45	Manic depressive	Circular; never keeping her balance between attacks; single
B60	25	Constitutional psychopath...	Two children before admission of whom one has since died; now six months pregnant; hypochondriacal
B61	42	Dementia praecox	Married; one child
B62	47	Unclassed	Single
B63	40	Dementia praecox	Single; at home; eccentric
B64	42	Unclassed	Married; no children
B65	55	Manic depressive	Attacks coalesced; two children
B66	52	Manic depressive	Married; no children; has been nervous and secluisve, but able to teach in the high school
B67	28	Unclassed	Married; one child before admission and one since
B68	48	Psychoneurosis	Married; three children; very well and capable between attacks
B69	29	Unclassed	Single; two attacks with marked improvement followed by death of an unknown cause at home

† Strecker, E. A.: Certain of the Clinical Aspects of "Late Catatonia" with a Report of Cases, Am. J. Insan. **74**: 185-220 (Oct.) 1917. A Psychosis of Seventeen Years' Duration with Recovery, J. A. M. A. **64**: 1151-1154, 1915.

Of the twenty-one deaths outside of the hospital, the causes were plainly evident in eleven on discharge from the hospital. Two others had nephritis and high blood pressure and died after two years at home; the type of illness seemed to be manic-depressive. Six patients died at home and two in other hospitals, from one to four years after discharge.

It should be remembered that all of the fifty-one deaths accounted for occurred with no marked improvement in the patient before death. A few others showed a tendency to improve before death and are classified elsewhere. The ages given on the chart and the diagnoses in Table 3 show that there is not much to expect in this group from new therapy. In the younger patients it is a matter of earlier treatment, of preventing infection (abortion, acute nephritis, syphilis) and diabetes, pellagra, exophthalmic goiter and of recognizing brain tumor—chiefly before the patient reaches the hospital.

This group of fifty-one patients, who had fatal illnesses, had had seventy-one children, of whom seven had died; there is a definite history of ten abortions. The average age is, of course, much greater than that of any other group. Many children were adults before the onset of the mothers' illness. No children were born after admission to this hospital.

UNIMPROVED

Group D, consisting of sixty-three cases, presents a sad monotony. Fifty-two are still under hospital care; one patient with a drug psychosis kept constantly to her addiction and was committed to several hospitals until the end of the five-year period when the chart was made—since then we have learned of her death from an overdose of chloroform. Patients D 30 and 31 could be followed for only three and one-half and four years.

As there are practically no other differences in the careers of these unimproved patients, details will be given grouped by diagnosis instead of on a chart.

TABLE 3.—DIAGNOSIS IN FIFTY-ONE CASES AND NUMBER OF DEATHS

Senile dementia	5
Psychosis with cerebral arteriosclerosis	8
Involutional melancholia	1
Involutional melancholia with tuberculosis	1
Involutional melancholia with diabetes	1
Multiple sclerosis	1
Psychosis with brain tumor	2
General paralysis (3); juvenile (1)	4
Psychosis with nephritis	1
Psychosis with nepritis, hepatitis, myelitis	1
Psychosis with pellagra	1
Psychosis with acute infection	8
Manic depressive	9
Dementia praecox	3
Unclassed	5
Total	51

Dementia praecox cases numbered thirty-four and all but three were in hospital after five years, three being under home care. The ages at admission were 18, 19, 20, 20, 25, 25, 25, 26, 27, 27, 28, 28, 28, 31, 32, 32, 33, 34, 35, 37, 37, 38, 38, 39, 43, 46, 46, 46, 47, 51, 54, 56 and 58. The ages tend to indicate late admission to the hospital, which is borne out by the histories. Naturally hospital residence has kept these patients from having children. Before admission they had had twenty-seven in all, one of whom was a musical prodigy, and another the apparent cause of the mother's chronic disease, the only living issue obtained by special precautions and caesarian section after three miscarriages.

Psychoses with senility and arteriosclerosis number four, all of them still under hospital care. The ages at admission were 71, 60, 72 and 62. There were no children.

Of manic-depressive patients, six remain essentially unimproved—three of mixed type with admission at the ages of 48, 50 and 52, one depressed admitted at 73 and two of manic type admitted at the ages of 43 and 58. None of these have had children since their admission as patients; they had had twenty before.

Two patients with epileptic dementia, admitted at the ages of 10 and 42, are now worse. The latter had had three children.

Four patients with drug psychoses progressed unfavorably, one of them taking an overdose, after the five-year period was charted. They were admitted at the ages of 30, 38, 38 and 46. Before admission one patient had had two children and two others, one each; they had none afterward. The fourth patient had no children and was divorced; it is not certain that she has had no children in the five-year period, but it is unlikely.

Three psychoses in psychopathic patients aged 29, 40 and 37 remain unchanged. There had been a record of one child (and several abortions) in this group.

One feebleminded girl is now in a home (aged 12).

TABLE 4.—DIAGNOSIS OF TWO HUNDRED AND FIFTY-ONE CASES

Manic depressive	82
Dementia praecox	47
Unclassed	42
Senile-arteriosclerosis	17
Psychoses with somatic disease	17
Involutional melancholia	14
Psychopathic inferiority	11
Drug psychoses	6
Psychoneuroses	5
General paralysis	4
Organic brain disease	3
Epileptic psychoses	2
Feebleminded	1
Total	251

A patient whose psychosis seemed closely associated with heart disease, whose blood pressure at the age of 40 was systolic 230 and diastolic 104, is still living in the department for mental diseases of the Philadelphia Hospital (single).

The condition of eight unclassified patients was left unchanged. The ages at admission were 31, 38, 63, 28, 47, 58, 22 and 51. Fourteen children were born to them before admission.

In this entire group of unimproved, the patients had sixty-eight children.

SUMMARY

These 251 patients were admitted five years ago in the ordinary course of hospital work. They were consecutive admissions, received on the average some time after the onset, and divided by diagnoses as indicated in Table 4.

Of this big group, sixty-eight patients have gone back to the community fully recovered, sixty-one have gone back to the community on a self-supporting basis, eight have shown marked improvement, in fifty-one the illness has progressed to death, and in sixty-three there

has been essentially no improvement. New therapy has its greatest opportunity in the second and fourth groups; it may stabilize those tottering patients who show a strong tendency to improve in any case, and anything it can do in the unfortunate last group will be clear and striking gain. The deaths may be lessened by gains rather in general medical knowledge than in psychiatry—by procedures against infections and for prolonging life. A point worth emphasis is that a new therapeutic procedure must show tremendous results to better the record made by the first group under the usual procedures of 1914.

TABLE 5.—RESULTS IN PATIENTS WITH SEVERAL CONTRASTING DISEASES

	Recovered	Recovered Minus	Died	Unimproved
Dementia praecox	1	9	3	34
Manic depressive	40	27	9	6
Involutional melancholia	6	5	3	..
Senile-arteriosclerosis	..	..	13	4
Unclassified	14	17	5	8

What happened to patients with several contrasting diseases is shown in Table 5. It speaks for itself. The dementia praecox patients stay; the only deaths occurred in three who were taken home. The senile-arteriosclerotic patients die, although we all know patients who seem to be exceptions. The manic-depressive death rate is not inconsiderable; deaths during excitement were always partly due to another disease. The unclassified, a group kept large in order to keep other groups pure, had an even distribution of results; most of them were unproductive, resistive patients with no signs of organic brain disease.

The total number of children born to these 251 patients was 286, of whom nineteen died, leaving 267, or over one to a patient, nine of whom were born since the admission of their mothers to this hospital.

Abstracts from Current Literature

GRASP REFLEX WITH BRAIN TUMOR. A CONTRIBUTION TO THE STUDY OF THE LOCALIZATION OF THE CENTER OF SEVERAL COMPLICATED REFLEX MOVEMENTS. W. PARRISIUS, Deutsch. Ztschr. Nervenheil. **67**:29, 1920.

Parrisius bases his observations on the following case: Frau K., 47 years of age, was first observed August, 1919. Her left arm became weaker, which annoyed her in agricultural work, and her gait more clumsy, owing to weakness in the left lower extremity. For some time she had been unable to let go of utensils which she grasped with her left hand. In September she fell twice in an attempt to get out of bed; following that she suffered with headaches.

The physical findings on admission to the clinic in October were: Strength in the left arm and leg was somewhat reduced; there was considerable spasm of the arm and leg musculature. On the left the periosteal and tendon reflexes in the arm and leg were increased, and there was a slight foot clonus; distinct Babinski and Oppenheim reactions were present. Only the upper abdominal reflexes were obtained (flaccid epigastrium, pregnancy). Perhaps the speech was somewhat indistinct. The left hand could be voluntarily closed into a fist only quite slowly, but if one lay in her hand an object such as a handle of a percussion hammer, or if one touched the surface of the hand quite gently, it closed immediately, while the thumb and index finger grasped quite energetically, like the claw of a crayfish. The patient was unable to open the tightly clenched fist.

Nine days later there was a slow, distinct improvement of the hemiplegic appearance; however, the left hand could not be closed voluntarily on command. At the slightest touch of the palm the phenomena described took place. The reaction seemed to be greatest if the forearm was previously supinated. Then simultaneously with the closure of the fist pronation occurred. If one pulled on a seized object, the fist closed more firmly. If the patient accidentally came in contact with the slightest object, then closure of the fist occurred as a reflex action. A sister frequently had to loosen the fingers from an object. Abduction, adduction, pronation and supination movements in the wrist were awkward, and combined purposeful movements, such as grasping the nose or ear, and other movements were awkwardly but correctly executed. In the legs no kind of apractic disturbances were demonstrable. After four weeks of treatment at the clinic there was slight improvement to all appearances, and the patient was discharged. In February, 1920, the patient appeared again in the clinic in substantially worse condition. There was a facial paresis on the left; paralysis of the left arm and leg and excessive spasms in both. The reflexes were greatly increased, the Babinski and Oppenheim signs were positive. The eyegrounds showed beginning papillitis, with swelling on both sides. The phenomenon of the left hand was no more demonstrable. Three weeks later the patent gave birth to a full time child, and five hours afterward died.

The pathologic findings at necropsy were: flattened convolutions and obliterated furrows. On the right half of the brain, in the middle of the gyrus frontalis superior, including the whole of the lobus paracentralis was a grayish-

red tumor, arching itself over the median plane and the corresponding part of the left half. This had grown firmly spread out posteriorly, with the falx cerebri. There was found also, on the left side of the falx, a similar tumor the size of a cherry stone. The tumor itself is described as being the size of a child's fist, and of a grayish-red, reddish speckled appearance, owing to the cut surfaces and lumina of numerous blood vessels. Anteriorly the tumor reached to the frontal region and occupied the whole gyrus frontalis superior and the gyrus cinguli, all but a thin strip. The corpus callosum was arched forward to the ventricle so that the anterior horn was severely compressed. The whole anterior central convolution was occupied, except a thin strip on its convexity. The severely flattened corpus callosum lay firmly on the compressed basal ganglions, especially the caudate nucleus, so that only a little space of the ventricle was present at the edge of the fornix; farther posteriorly, also, the gyrus centralis posterior was atrophied on the convexity, all except a thin layer. The thalamus was strongly compressed, the ventricle showing only a slight crescentric lumen. Behind the gyrus centralis posterior the tumor ended, the flattened pole of the egg-shaped structure reaching to the parietal region. Here the lateral ventricles were widened, particularly the posterior horns. The final anatomic diagnosis as given was—large endothelioma of the dura mater.

The author raises the question as to what we are trying to learn from the case described in the foregoing. He classes this as an apractic disturbance of the left hand in the sense of the motor or ideokinetic apraxia of Liepmann. The author cites similar cases from the literature, as, for instance, that of Liepmann, whose patient could not close his hand on command, but in whom the closure succeeded when an object was placed in the hand. The same patient on command could not perform the act of buttoning, but if the hand was placed on the button hole, he smoothly accomplished this act. In all the cases cited voluntary movement of various muscles was not possible, but on being stimulated they could promptly perform their function. Liepmann concluded that the stereotype repeated purposeful movements, such as walking, winking, fist making, to seize or hold firmly an object, as special performances of the sensory-motor mechanism could be satisfactorily performed subcortically, perhaps also spinally, although their peculiar nervous substratum is found cortically in the cortical fields of the various limbs in the form of kinetic memories. This controlling influence of the higher centers as, for instance, the optic and acoustic, is not necessary, as they are performed to some extent through cortical "short circuits" within the sensomotorium dependent on tactile irritation. Granted that the senso-motorium may be completely separated from these higher centers, then the ideatory and the kinetic memories are torn asunder; it therefore follows that the closure into a fist may still result on tactile stimulation as an independent action of the senso-motorium, but not without the proper stimulus or as a repetition (imitation) of an act already performed.

The author states that his case was different from that of Liepmann and others, not only in the fact that a closure of the hand was possible, if an object touched the palm, but that it followed purely reflexly, the patient being unable to inhibit it. This is, according to the definition of Munch-Petersen, a true skin reflex; that is, an involuntary movement which originates through stimulation of the skin and continues; that the skin reflexes are purposeful movements which have as their purpose to remove unpleasant or destructive stimuli from the irritated portion of the body. In this case not only the palm was closed reflexly but also the thumb became active in a very important degree, and produced throughout the impression of grasping or of a claw; therefore the reflex is

fitly named the grasp or claw reflex. In the literature the authors found few accounts of similar observations. A palmar reflex, which consists in a palmar flexion of the fingers with irritation of the palm, was mentioned more often than the grasp reflex. Lewandowsky, in mentioning it quite briefly, places the anatomic substratum in the spinal cord—cervical 7 to dorsal 1.

The author seems to be undecided whether the palmar reflex is a preliminary step or an attenuated form of the grasp reflex, or whether it indicates something entirely different. Lewandowsky has observed the grasp reflex in anencephalic patients and Janischewsky has reported two cases almost identical to that of the author. Janischewsky localizes the process in the opposite hemisphere beneath the cortex in the region of the gyrus centralis anterior, and names the process "reflex de préhension."

Strümpell has advanced the following theory concering skin reflexes: These reflexes, even if now appearing to be practically useless, have been in their phylogenetic past most probably attack and defensive movements; they have passed through numerous generations until they have now taken their stereotyped form with a corresponding laying down of anatomic paths. The higher the organization the more they subside and are replaced by voluntary movements. It is therefore no accident that the skin reflexes in the upper extremities are far more unusual to obtain than in the lower. This makes it clear why some reflex phenomena observed in idiots otherwise only appear in infants. According to Doflein, the tendency to grasp spasmodically offered objects in infants is based on atavistic phenomena, and following Strümpell's theory, the loss of the reflex movements is explicable accordingly as useful voluntary movements are acquired.

The author then seeks to localize the site of such reflexes in the brain, since it has been conceded by Munch-Petersen and others, based on recent investigation, that the localization of the reflex center in the cord may have become untenable. Speaking of Oppenheim's feed-reflex, Fürnrohr and others have found a definite area in the cortex whose stimulation produces the characteristic reflex. However, they are led to believe that the real center is in the optic thalamus and that the function of the cortex is simply regulatory and inhibitory. Janischewsky places also the center for the grasp reflex in the subcortical ganglions, probably in the optic thalamus, and he writes that the grasp reflex may be made use of as a diagnostic sign for lesions of the frontal lobe above the subcortical centers (he classes the gyrus centralis anterior with the frontal lobe).

The author concludes that the question of the site of the center for such a reflex is still not definitely decided. This reflex belongs in the series of those which are observed in infancy, but later disappear. Probably the subcortical ganglions would have to come into consideration, and as they are controlled by the cortex, the appearance of this reflex at a later age would, under these hypotheses, speak for a disturbance of the paths from the cortex to the subcortical ganglions.

WAGENHALS, Columbus, Ohio

CHANGES IN THE BRAIN IN LETHARGIC ENCEPHALITIS.

CHARLES B. DUNLAP, State Hosp. Quart. **6**:311 (May), 1921.

According to my present experience, lethargic encephalitis is a so-called inflammatory disease with a cellular exudate. This exudate is likely to consist mainly of lymphoid cells with fewer plasma cells or sometimes none, but if any one expects to make his diagnosis on the make-up of the exudate alone he

will be disappointed. It is the location of the cellular exudate rather than its character that helps in diagnosis.

In the so-called typical cases of lethargic encephalitis the exudate is seen abundantly in the brain stem, particularly in the midbrain, where the nuclei of the oculomotor nerves are found; it is therefore not strange that paralyses of the eye muscles often occur in lethargic encephalitis. A peculiarity of this exudate is that often it is not confined within the outer sheaths of the blood vessels, as in general paralysis, but, with the vessels as centers of greatest intensity, the exudate is scattered about through the nervous tissues.

The nervous tissues into which the exudate escapes in encephalitis, including the nerve cells, do not seem to be profoundly altered by it, and we have not noticed any marked changes in the neuroglia cells, although occasionally some spider cell reaction and some increase in satellites may be seen.

Another common feature in these cases is great engorgement of the blood vessels, usually with slight local escape of blood into the tissues, so that hemorrhages, of small size, are frequent in lethargic encephalitis, a condition usually not seen in cases of general paralysis or syphilis.

So in the more typical cases of lethargic encephalitis we have, among the cardinal signs of inflammation, redness, swelling and diffuse exudate, especially in the brain stem, with comparatively little of this in the cerebral hemispheres or cerebellar hemispheres.

In the less typical cases, diagnosis with the microscope is much more difficult. In some of these cases we find little exudate in the brain stem, merely a few lymphoid cells around a small number of blood vessels and a few small hemorrhages here and there. The same sort of thing is also found in the gray matter of the cerebral hemispheres, and in the pia mater small bunches or rows of lymphoid cells are seen here and there. The more we looked the more we found in these less typical cases; sometimes, in a case of this kind, a massive lymphoid exudate might suddenly and unexpectedly appear in the microscopic field. For example, in the caudate nucleus of one case a large amount of exudate was found in the walls of only a few blood vessels.

Another rather striking thing throughout the series of cases (both typical and atypical) was a marked swelling of the endothelial cells of the blood vessels and often an apparent increase in number of such cells. These endothelial cells have the property of taking up and disposing of injurious substances and, especially when they get free, they are often actively phagocytic.

In one doubtful case, there were, in all parts of the cortex, small foci of such free endothelial cells around small blood vessels, and the presence of mitotic figures in some of these cells showed that they were actively subdividing. Whether rightly or wrongly, we interpret this as meaning that not only the cells of the exudate, but also the endothelium is active against the effects of the invading foreign material, whatever it may be (globoid organism of Loewe and Strauss?), in lethargic encephalitis.

In trying to differentiate other toxic and infectious processes from what we have called the more diffuse processes as seen in lethargic encephalities, it would seem wise to use both history and microscope, make a reserved differential diagnosis and then leave a loophole for escape.

Except in the most typical cases of lethargic encephalitis, a diagnosis based on histology alone is not safe. There are no absolutely specific pathognomonic features to depend on.

POLLOCK, Albany.

PERSONAL RESULTS IN THE INTRASPINAL TREATMENT OF NEUROSYPHILIS. B. Rodriguez, Rev. Neurol. **27**:439 (May) 1920.

Rodriguez reports a series of fourteen cases of neurosyphilis in which intraspinal treatment was preceded and combined with vigorous constitutional therapy—intravenous neo-arsphenamin associated with mercurials and potassium iodid. Specifically, in the matter of intraspinal therapy the author utilized both mercurialized serum (in vitro) in dilution of 10 c.c. of serum to 5 mg. of mercuric chlorid and neo-arsphenaminized serum (in vitro) in dilution of 10 c.c. of serum to 5 mg. of neo-arsphenamin (Billon), preferring autoserums where possible, as inducing a minimum constitutional reaction on the part of the patient. It might be mentioned that the paresis cases were noted to be definitely hyporeactional, from this point of view, as compared with the other types of neurosyphilis. The dosage varied, progressively, from 1.5 to 5 mg. in the case of mercuric chlorid and from 2.5 to 5 mg. in the case of neo-arsphenamin, with rest periods of from two to eight weeks.

Comprising the author's series were five cases of general paresis, two of tabes dorsalis, one "cerebral" syphilis, one of cerebrospinal syphilis, four of "spinal" syphilis and one of heredosyphilis with eighth nerve involvement.

Of the first group, in which from two to four subdural injections of mercurialized serum were administered, ranging in dosage from 1.3 to 1.6 mg. at intervals of from two to four weeks, four cases showed transient improvement clinically, with no evidence of amelioration in the fifth, and in all but one, the spinal fluid Wassermann reaction and the colloidal gold curves became somewhat less positive. Of the two tabetics the first, with negative spinal fluid Wassermann and Nonne-Apelt reactions and a colloidal gold curve of 01100000000, showed gradual, but marked improvement after an injection of 1.3 mg. of mercurialized serum, and the second, with feebly positive spinal fluid Wassermann and Nonne-Apelt reaction and with a colloidal gold curve of 11210000000, in which four injections of autoneo-arsphenaminized serum were administered in doses of 2.5, 3.5, 4.5 and 5 mg. at intervals of one and a half months, showed only slight clinical improvement, with some reduction in the colloidal gold curve. The case of "cerebral" syphilis with negative spinal fluid Wassermann and Nonne-Apelt reaction and a colloidal gold curve of 11222300000, showed prompt clinical improvement after one injection of 3.5 mg. of automercurialized serum. In the case of the patient with cerebrospinal syphilis in which the spinal fluid Wassermann reaction was negative, the Nonne-Apelt reaction feebly positive and the colloidal gold curve 11210000000, the administration of three injections of autoneo-arsphenaminized serum in dosages of 2.5, 3.5 and 4 mg. at an interval of one and one-half months, showed slight clinical improvement, negative Nonne-Apelt reaction and slight lowering of the gold curve. In the four "spinal" patients (apparently Erb type) who received two to five injections in doses ranging from 1.3 to 5 mg., of both mercurialized and neo-arsphenaminized serum, there was noted definite clinical improvement and in the heredosyphilitic case, with negative Nonne-Apelt and Wassermann reactions and negative colloidal gold curve, two subdural injections of 1.3 and 2.6 mg. of mercurialized serum were administered at an interval of three weeks with marked clinical improvement. No definite statement is made in these cases as to the permanency of the improvement noted.

In concluding, the author emphasizes the necessity for early, vigorous and persistent, combined treatment in all cases of neurosyphilis, particularly in the paretic and tabetic types, which he found to be, therapeutically, by far the least amenable.

RAPHAEL, Ann Arbor, Mich.

PATHOGENESIS OF CATATONIC STUPOR. H. BERGER, München. med. Wchnschr. **68**:448 (April 15), 1921.

Twenty years ago Berger observed that a subcutaneous injection of cocain in a severe case of catatonic stupor produced a transient disappearance of the mental condition. In the present article he presents the results obtained in a study of eleven cases.

In this work the author used from 0.025 to 0.05 gm. of cocain hydrochlorid. Eight of the patients showed decided reactions; three showed no change. The three patients showing no reaction had long standing cases of dementia praecox, while the eight who reacted had rather acute cases of dementia praecox.

In the eight patients the reaction was temporary and frequently the period of mental change lasted only from one to two hours. As a therapeutic measure cocain offers no hope.

Cocain, like other poisons that act on the central nervous system, cannot cause qualitative changes in the central functions, only a heightening or lowering of the normal activity of these centers.

The animal experimentations as carried out by Uyolino, Mosso, and others, have shown that cocain injected in small doses produces stimulation of the cerebral cortex. In man the first noticeable action is a sensation of warmth and a pronounced feeling of well-being. The psychic phenomena are not entirely clear, however. The author used cocain in several cases of manic-depressive insanity of the depressed type and found a heightened psychic activity; thus their time reactions for simple mathematical equations was markedly increased. In one case the average reaction time was 24.9 seconds, while following an injection of cocain the reaction time was increased to 8.8 seconds.

The author does not believe in a psychologic motivating factor in the production of stupor. He believes that catatonic stupor, as a rule, is a primary stupor, so termed by Ziehen, and that it does not disappear by means of psychic influences. Berger does not accept the view of complex formation in stupor as held by Jung.

In the stupor of dementia praecox metabolism is impaired, as are other physiologic functions. The causes for the changes are not easily explained, but, according to Ziehen and others, are due to a marked cortical inhibition. Reichardt believes one must consider a physio-chemical change in the colloidal brain substance, while Bleuler in several cases of stupor has found edema of the membranes with brain swelling.

The author assumes that cocain has a stimulating action on the cerebral cortex. In cases of primary stupor the cortical activity is greatly diminished, and by the injection of cocain there is a stimulation of the "dissimulation processes" with subsequent increase of cerebral function, and as long as the cocain action continues the stupor disappears, to return with the disappearance of the cocain stimulation.

Why the cerebral function becomes impaired is another question. Certainly in the long standing cases organic changes do occur, as is also indicated by their failure to react to cocain stimulation. In agreement with the anatomicopathologic findings, one may assume that changes in the second and third layers of the cortex do not permit the proper function of these cells to take place.

Berger believes that a primary catatonic stupor is an organic syndrome which is due to inhibition or diminished activity of the cerebral cortex. He concludes with the remark that he cannot accept the psychic genesis of catatonic stupor, as a matter of fact of dementia praecox.

MOERSCH, Rochester, Minn.

SIMULTANEOUS MULTIPLE CLONI IN EPIDEMIC ENCEPHALOMYELITIS. G. DAGNINI, Gior. di clin. Med., Pt. 7-8 (July-August), 1920.

By simultaneous multiple cloni the author refers to the manifold manifestations of motor irritation which under the form of involuntary muscular twitching, of different amplitude, localized or disseminated, regular or arythmical, have been observed during the recent epidemic of lethargic encephalitis. He has made a study of these simultaneous clonic contractions in several muscular groups whose action in some cases is asynergic and antagonistic.

In one of the cases tracings of the muscular contractions were taken and compared two by two in the following order: left latissimus dorsi and flexors of the head, right pectoralis major and left flexor carpi radialis, left flexor carpi radialis and flexors of the head, left extensor carpi radialis longus and right pectoralis major, right pectoralis major and elevators of the left shoulder, left scalenus anterior and right rectus abdominis, left flexor carpi radialis and right rectus abdominis. Perfect synchronism of the muscular contractions was demonstrated in every one of the muscles examined, including the respiratory muscles. The contractions ceased during sleep, and could not be influenced by the patient's will. Emotions, suggestion, peripheral stimulation or bromids did not modify the intensity, the frequency, the rhythm and the localization of the cloni. Synchronism was not accidental.

The author points out that in these cases, unlike those of epileptic seizures, of tic convulsif and also of chorea, in which the synergic movements originate from a contemporaneous stimulus acting on centers having reciprocal anatomic and functional connections, it is difficult to interpret the appearance of simultaneous clonic contractions in muscular groups whose centers are not in direct relationship anatomically or functionally.

What cortical, subcortical, spinal, radicular and peripheral relationship exists —the author asks—among so distant and functionally different muscles, whose centers react contemporaneously to the same stimulus? Supposing that an abnormal stimulus, starting from a slight lesion in the rolandic zone, caused contractions in two muscles of the forearm, why were not the other nearby centers for the upper left extremity involved, whereas more distant centers (for the elevators of the shoulder, latissimus dorsi, scaleni, pectorales, diaphragma) and even contralateral centers were irritated? If the phenomenon were of spinal origin, why did cellular groups belonging to various segments from the cervical cord (flexors of the head) to the dorsal and lumbar sections (rectus abdominis) react simultaneously to a stimulus without the near and the intermediate zones being involved?

The author propends in favor of a spinal origin. Small symmetrical lesions of the anterior horn cells may explain better the phenomenon, not that symmetrical movements cannot originate from unilateral lesions of the brain, but because the succession of the contractions suggests the existence of two distinct lesions localized in homologous seats. The stimulus must have different effects according to the degree of excitability of the cellular elements on which the

identical stimulation is successively exerted. Probably a condition of increased excitability of each clonogenous cell plays an important rôle in circumscribing within given zones the effects of a stimulation which may also irradiate elsewhere, if the recepting cellular elements have not reached a sufficiently high reacting power.

There may be an analogy with the systemic lesions of the spinal cord, in which probably the bilateral and uniform morbid localization is dependent on chemical affinity of given poisons for groups of cells or fibers symmetrically disposed and equally subject to the same pathogenetic cause.

NACCARATI, New York

A STUDY OF THE SPINAL FLUID IN ONE THOUSAND EIGHT HUNDRED AND SIXTY-NINE CASES OF SYPHILIS IN ALL STAGES. U. J. WILE and C. H. MARSHALL. Arch. Derm. & Syph. **3**:272 (March), 1921.

The authors quote a previous communication of their own by restating that the invasion of the nervous system in syphilis occurs extremely early—even seriously—without symptoms, and undetected except by means of spinal fluid examinations. They believe that cerebrospinal involvement, when occurring, does so within the first few months of the infection. The conclusions based on a study of the 1,869 positive cases herein reported are that if the nervous system is not involved early in the disease it is not often involved later. Furthermore, they view the high percentage of positive findings in primary and secondary syphilis as in the nature of a meningeal roseola and transitory in character. On the other hand, a certain percentage of cases is found in which the cerebrospinal involvement is considerable but shows few signs or symptoms; these cases are in the latent period and may become symptomatic later. In the tables accompanying the text it is shown that there is considerable parallelism in the percentage of positive findings.

PERCENTAGE OF POSITIVE FINDINGS

Type of Syphilitic Involvement	Number of Cases	Percentage Increased Albumin and Globulin	Percentage Increased Cells	Percentage Positive Wassermann Reaction
Congenital	53	13.2	26.4	16.9
Primary	236	19.0	16.1	5.9
Secondary	508	35.1	34.0	26.5
Tertiary	155	26.4	25.1	30.9
Latent	568	27.1	28.6	27.9
Diffuse cerebrospinal	178	97.2	88.2	87.6
Tabes	109	98.1	93.5	93.5
Paresis	49	95.9	95.9	98.9
Taboparesis	13	100.0	100.0	100.0

By reference to the consolidated table, it is noticed that of nervous system involvement, cerebrospinal syphilis is lowest in uniformity of positive findings. The authors deal briefly with the frequency of association of neurosyphilis with syphilitic alopecia, iritis, leukoderma colli and late syphilids of the skin. There is a high percentage of asymptomatic neurosyphilis cases during the period of latency.

The paper is very interesting from the statistical point of view, and it is regretted that the colloidal gold curves could not have been made in each case to complete the data.

PATTEN, Philadelphia

SOME PRACTICAL POINTS IN THE ORGANIZATION OF TREATMENT OF SYPHILIS IN A STATE HOSPITAL. AARON J. ROSANOFF, State Hosp. Quart. **6**:319 (May), 1921.

The cases of syphilis which are encountered among state hospital admissions may be classified as follows:

Systemic syphilis—latent; active. Neurosyphilis—mesoblastic (cerebral) syphilis); parenchymatous (general paralysis).

As regards latent systemic syphilis, the main point is that it can be brought to light only by means of routine Wassermann tests made on all patients admitted to the hospital. There is, as a rule, no etiologic connection between the syphilis and the mental disorder in such cases. The patients should, however, receive antisyphilitic treatment on general principles, and, in event of parole, provision should be made for continuance of such treatment extramurally.

As regards cases of active systemic syphilis, it need hardly be said that these patients are in even more urgent need of prompt, vigorous and persevering treatment, although in such cases, too, there is no etiologic connection between the syphilis and the mental disorder.

In cases of mesoblastic invasion, we find, as the most significant of the recently discovered facts (1) that such invasion occurs in a high percentage of cases early in the course of the infection, namely, in the primary and secondary stages, and (2) that it may exist without giving rise to nervous or mental symptoms, or may manifest itself by slight, vague, or transient symptoms, so that there is danger of its being overlooked.

The practical bearing of these facts is that all patients with systemic syphilis, latent or active, should be, at some time in the course of their treatment, investigated by lumbar puncture, in order to dispose of the question of possible invasion of the central nervous system; and that no patient with syphilis can be discharged as cured until not only the clinical and blood serum findings have been rendered persistently negative but also the spinal fluid findings.

We now come to the consideration of neurosyphilis caused by parenchymatous invasion, i. e., general paralysis. In this connection the question is perhaps to be formulated as follows: Considering all past experience, which is discouraging, should such patients receive antisyphilitic treatment? In my opinion, the answer must be in the affirmative.

There has been much discussion as to the choice between arsphenamin and neo-arsphenamin. The question presents for consideration three distinct elements: (1) trypanocidal power, (2) toxicity, and (3) convenience in administration.

Experimental investigations recently made by Schamberg and his associates in the Dermatological Research Laboratories of Philadelphia have shown that arsphenamin is one and three-fourths times more curative than neo-arsphenamin, but is at the same time two and one-half times more toxic. Accordingly, it is possible to choose for neo-arsphenamin such dosage as to secure greater curative and less toxic effect than by means of arsphenamin.

The technic of administering neo-arsphenamin is by far the simpler for the physician and the less uncomfortable for the patient. The ampule is opened by filing and breaking off the pointed end; the neo-arsphenamin is dissolved in the ampule with about 5 c.c. of freshly distilled water at room temperature; and the solution is slowly injected intravenously with a 10 c.c. glass syringe. A dose of 0.9 gm. should be given in not less than 10 c.c. of water. The ampules in which the neo-arsphenamin is furnished generally have a capacity of only

a little over 5 c.c., but more water can be drawn into the syringe to dilute the solution before it is injected. Higher degrees of concentration, especially if injected too rapidly, cause hemolysis, which gives rise, within twenty-four hours, to a reaction characterized by chills, nausea, vomiting, headache, pains in the arms and legs, some general prostration and occasionally slight elevation of temperature.

POLLOCK, Albany

THE DIFFERENTIAL DIAGNOSIS BETWEEN MYOCLONIC UREMIA AND EPIDEMIC ENCEPHALITIS. HENRI ROGER and ANDRÉ CHAIX, Presse méd. **29**:461 (June 11), 1921.

Two cases of uremia with myoclonic symptoms are reported in detail and compared with others in the literature. Some fairly definite cases of myoclonic epidemic encephalitis have shown pathologic kidneys at necropsy, so that the symptom in question could not be ascribed to either cause exclusively. Clinical differentiation may also be difficult. Epidemic encephalitis is responsible for the majority of such cases, while toxic conditions other than uremia can also give rise to a similar syndrome.

Uremia should show characteristic clinical signs, such as vomiting and epistaxis, and give a history suggestive of renal insufficiency; but without these the absence of fever and pains when uremia or encephalitis is suspected is in favor of the former. Epidemic encephalitis of the myoclonic type nearly always causes a febrile reaction, at least at the beginning, while pains are usually noticeable and often severe. One must remember that hiccups and lethargy are uremic as well as encephalitic signs.

The cases of myoclonic uremia were characterized by sharp contractions and twitches with little movement at the joints, attacking limbs, face and abdomen indiscriminately. These contractions were sometimes choreiform. Reflexes were usually exaggerated, with clonus.

A lumbar puncture yielding evidence of meningeal irritation does not necessarily exclude uremia. Neither does a slightly increased urea content of cerebrospinal fluid and of blood exclude encephalitis. It has been claimed that histopathology could furnish positive differentiating evidence: that degenerative or inflammatory changes found in encephalitis would not be found in uremia. These writers, on the contrary, expect that central nervous lesions will be demonstrated.

Uremia with generalized myoclonic contractions offers a bad prognosis; if the symptoms are localized the prognosis will be better. Treatment is the same as for any other form of uremia.

Besides myoclonic uremia, four other nervous types are described: eclamptic, jacksonian, tetanic (disputed by some authors) and ataxic.

HUDDLESON, New York

ABNORMAL LOCALIZATION OF TETANY IN THE ADULT. VICTOR CORDIER and AUGUSTE GONNET, Rev. Neurol. **27**:244 (March) 1920.

Cordier and Gonnet report a case peculiar, or of the fruste type, in that only one leg was involved, but that typically, directing attention, at the same time, to the scant recognition accorded such apparently aberrant forms in the literature, in spite of their comparative frequency clinically.

The case was that of a man, aged 67, a cabinet maker, who complained of shortness of breath, noted for five years past, and painful cramp-like attacks

affecting the left leg, first noted about two years previously. In regard to the latter it was reported that the attacks occurred at intervals of from about eight to fifteen days and could be controlled only by continued rest in bed in the extension position. There was a history also of similar attacks affecting the fingers six years previously as the result of which the patient was no longer able to use his joiner tools. There was no history of alcoholism and no clinical evidence of syphilis.

Physical examination showed well marked emphysema, chronic bronchitis and a blood pressure of (systolic) 160, (diastolic) 80, but was otherwise essentially negative.

Neurologic examination, during one of the "leg attacks" showed the anterior crural and quadriceps femoris muscle groups, of the left leg, to be in tonic contracture with the toes, particularly the hallux, in hyperextension and the knee somewhat elevated. The other muscle units were not involved. This contraction phase was associated with excruciating pain of a local and cramp-like nature. Relaxation occurred in approximately ten seconds and was succeeded by another phase of contraction which lasted about four or five seconds and so on for two minutes. Stimulation of the muscle elements involved was found to precipitate a typical contracture phase, but no change was noted on stimulation of the other muscle groups. Electrical examination showed hyperirritability of the tibial and common peroneal trunks. Neurologic examination was otherwise completely negative.

Etiologically the authors seem inclined to discount the importance of paratyphoid disturbance in many cases of tetany, so-called, and on the basis of the case presented suggest the possible significance—direct or indirect—of occupational posture-tensions.

RAPHAEL, Ann Arbor, Mich.

BRAIN AND THYROID FUNCTION, C. CENI, Riv. sper. di reniat. **44**: 243-286 (July), 1920.

Ceni has studied the effect of partial and total removal of the brain on the function of the thyroid gland.

In lower vertebrates (*Chelonia*) no relation was found to exist between the forebrain and the thyroid function. The total extirpation of the brain produced neither immediate nor later changes in the thyroid.

In birds (fowl and pigeon) decerebration and hemidecerebration was followed by diffuse colloid degenerative atrophy within twenty days from the operation.

In the same animals, dead or killed from thirty-five days to three years after the operation, the thyroid alterations were more pronounced; they involved all the thyroid structures (colloid, alveoli, septums, epithelium, vessels), assuming the character of diffuse parenchymatous and interstitial hypertrophy.

In fowls no definite relation was found to exist between the states of atrophy and of hypertrophy of the thyroid gland and the general development of the body and of the external sexual characters. No constant relation was found between the hypofunction and the hyperfunction of the thyroid gland and the hypofunction and hyperfunction of the sex glands.

In dogs the removal of the cortex of one cerebral lobe or of the surface of one cerebral hemisphere generates thyroid alterations similar to those found in birds, namely, diffuse colloidal hypertrophy, which may also reach the degree of parenchymatous hypertrophy. These phenomena, likewise in birds, are not constant, but are frequent and vary in degrees in divers subjects.

The general conclusions which the author draws from his experiments are:

1. In vertebrates exist superior thyroid centers, which in birds are disseminated in the mass of the whole forebrain, especially in the striate body, and in mammals are distributed in the entire cerebral cortex.

2. The superior thyroid centers have a trophodynamic inhibitory function, which, in opposition to the accelerator function of the genetic centers, act by preventing an excessive function of the peripheral organ, by regulating the circulation and by maintaining in normal proportions the trophic processes of the different elements, especially of the parenchymatous ones.

The author points out that the results of his experiments call for a revision of many clinical problems, especially in neuropsychiatry, in view of the present tendency on the part of clinicians to interpret any alteration of the thyroid gland as a cause rather than as an effect of the clinical picture.

NACCARATI, New York

REGARDING A NEW SUPERFICIAL REFLEX—THE PALMO-MENTAL REFLEX. G. MARINESCO and A. RADOVICI, Rev. Neurol. **27**:237 (March) 1920.

The authors describe an additional reflex of the cutaneous series for which they propose the designation palmo-mental reflex.

This reflex, which the authors have found to be particularly characteristic in pyramidal tract lesions, especially when bilateral, consists in a slow and enduring contraction of the superficial chin muscles (triangularis, quadratus, labii inferioris and transversus menti) ipsolaterally, following stimulation of the palmothenar area. Care must be taken in eliciting this response that the chin muscles be relaxed and the mouth held slightly opened; gross posture is of no primary importance as the reflex may be elicited equally well with the patient sitting or in lateral decubitus. It may be remarked that in pathologic cases this mental response may be secured from the proper stimulation of practically any sensory area, but seems most delicate, in the authors' experience, on stimulation of the thenar surfaces as described.

This response was also noted in 50 per cent. of a series of 300 normal subjects (particularly in infants), but differed from the reaction determined in pathologic cases in that the contraction was definitely quicker, of shorter duration and that the response was noted only after stimulation of the palmothenar surfaces and did not follow excitation of other sensory areas.

This reflex is found to be abolished in the presence of first motor or sensory neuron lesions, thus being of interest in the differentiation of functional and organic disorders. The mechanism of response is not wholly clear although the authors feel it to be mediated, indirectly, by intercalated neurons through the lateral columns and thence, by collaterals, to the facial nuclei, ipsolaterally.

RAPHAEL, Ann Arbor, Mich.

THE SUBSTANTIA NIGRA: A BRIEF COMPARATIVE ANATOMICAL STUDY. I. S. WECHSLER, Neurol. Bull. **3**:130-134, 1921.

The author reviews the literature on the substantia nigra and gives the results of his own study of serial sections of seven simians, two other animals and man. He states that the structure is about the same in all mammals, though it is best developed in man. It is not well defined and cannot be sharply

delimited from the tegmentum, internal geniculate body, pontile nuclei or the hypothalamic region. It contains four groups of cells. The fiber connections are not thoroughly worked out, but as nearly as can be determined, it is connected with the frontal, temporal and central cortex, with the caudate and lenticular nuclei, the internal geniculate body and the spinal cord. Whether these fibers are centripetal, centrifugal or both, has not been definitely decided. The substantia has not been studied in birds, and is said not to exist in the lower vertebrates. Bauer believes that it was originally an undifferentiated structure and that its cells have developed from the central gray and moved ventrally, which explains the reason why some of the cells are found in the lemniscus and reticular substance. Its function is summarized in a paragraph from Bauer, as follows: "Just as the numerous cells grouped throughout the brain-stem and conceived by Edinger as the nucleus motorius tegmenti are under the influence of the cerebellum, so the cells of the substantia nigra are for the purpose of coordination under the influence of the cortex. The cells carry impulses from cerebral centers, from the cortex or corpus striatum, directly or through association cells of the tegmentum, to various efferent cell complexes. The coordination of chewing and swallowing is only one of the numerous associational and coordinative functions of the substantia nigra."

HOWE, New York

PLACENTAL TRANSMISSION OF EPIDEMIC ENCEPHALITIS. R. MERCIER, in collaboration with ANDRIEUX and BONNAUD, Bull. de l'Acad. de med. **85**:625 (May 31), 1921.

A young woman, aged 28, twenty-two days before delivery of a child, developed epidemic encephalitis, with pains and myoclonia at the onset and subsequently diplopia, abolition of reflexes and delirium.

The spinal fluid was clear, without bacteria or lymphocytosis. There were appreciable traces of albumin and of sugar. The Wassermann test was negative.

At labor, though dilatation occurred, uterine contractions were deficient and delivery by forceps was necessarily resorted to.

There was no subsequent uterine infection. However, the labor appeared to act as a traumatic shock and greatly intensified the encephalitic symptoms, and the mother died in coma on the fortieth day.

The infant, who was from the beginning separated from the mother, showed myoclonic movements of the head and upper extremities, attributed to an encephalitis. These began in the second week of life and gradually disappeared over a period of three weeks. This condition was not accompanied by fever. Residuals in the child have been a certain nervous excitability and an occasional convergent squint. Otherwise, the child thrives.

These clinical facts are reported as well proving the transmission of epidemic encephalitis by the placenta.

DAVIS, New York

ISOLATED NEURITIS OF THE MOTOR BRANCH OF THE TRIGEMINAL NERVE. K. H. KRABBE, Rev. Neurol. **27**:241 (March) 1920.

Krabbe, directing attention to the rarity of isolated neuritis of the motor component of the trigeminal nerve, makes report of a case in which difficulty in opening the mouth and in mastication was noted several days after the onset of what appeared clinically as a typical attack of influenza with bronchitic features.

The systemic condition apparently subsided speedily and uneventfully, but the neurologic disturbance was noted to persist in diminishing progression for some months, localizing principally to the right with some flattening of the face on this side.

On examination by the author, ten months after the disturbance had first been noted, there was remarked pronounced atrophy of the right temporal and masseter muscles with marked weakness in the right bite. There was also indication of paralysis of the right internal pterygoid as it was impossible for the patient to draw the mouth to that side. The right corneal and masseter reflexes were found to be diminished, but no frank sensory deviation could be determined and no disturbance could be discovered in the fields of the other cranial nerves.

The author makes no mention as to the ultimate outcome in this case.

RAPHAEL, Ann Arbor, Mich.

THE MENTAL CLINIC AND THE COMMUNITY. EVERETT S. ELWOOD, State Hosp. Quart. **6**:381 (May), 1921.

The establishment of after-care work at the Manhattan State Hospital was the first definite move in this direction in the state of New York. This work has grown so that the state hospitals now have a total of twenty-three social workers and forty-one clinics. As the people become more and more acquainted with it their prejudice and superstition against all forms of insanity gradually disappear.

The work of the mental clinics is greatly appreciated by most of the communities in which they function. During the last year we have received requests from several cities for the establishment of clinics under the guidance of state hospital physicians. The dean of a prominent medical college recently asked what he could do to facilitate the organization of this good work in his city. Public school departments are particularly anxious to receive the help that clinics afford in the diagnosis and treatment of borderline cases developing among schoolchildren.

The time is probably not far distant when each state hospital will have to have an extra physician on its staff to attend to this field work, for the assignments of duties within the institutions are in most instances sufficient to demand the whole time and energy of the medical staff.

Such a service to the community should have the adequate support not only of the state hospitals themselves and the various social forces in the different communities but also from the appropriating authorities of the state. It is not only a saving in dollars and cents to keep a patient sufficiently balanced to forego the necessity of hospital commitment, but it is a great saving in the welfare and happiness of the individual patient and his family.

POLLOCK, Albany.

CLINICAL AND ANATOMOPATHOLOGIC CONTRIBUTION TO THE STUDY OF HEREDITARY ATAXIA. G. MINGAZZINI and F. GIANNULI, Gior. d. r. Accad. di med. di Torino, 1919.

The patient who forms the basis for this anatomopathologic study was suffering from spinocerebellar ataxia. She had been for many years under the author's observation. The interest in this case is increased by the fact that

Mingazzini had studied in the same manner a brother of the patient several years ago. The conclusions reached by the authors in this research confirm the previous work of Mingazzini.

The present investigation has been extensively and accurately carried out with serial cuts of the brain and of the spinal cord; the latter has been studied with greater riches of particulars of the degenerated areas. There are thirty-four figures in the text.

One of the conclusions confirms the pseudosystemic type of the degenerative processes in this disease.

Another important conclusion regards the behavior of the glia. The authors demonstrate that glial alterations are found, not only in the spinal cord, but also in the cerebrum and in the cortex; such alterations follow the same phases and show the same characteristics observed in cases of disseminated sclerosis of the brain.

Finally, the authors, basing their assumptions on their histologic findings and on those of previous investigators, maintain that cerebellar ataxia is not a disease limited only to the cord and cerebellum, but that it represents a morbid condition affecting the whole spino-cerebro-cerebellum system.

The thesis advanced by the Roman school has been emphasized in one of Mingazzini's recent papers, in which he reiterates the fact that spinocerebellar aplasias, as well as some other diseases hitherto classified as purely spinal, are to be considered as cerebrospinal diseases.

NACCARATI, New York

NEW RESEARCHES ON THE MORPHOLOGY AND BEHAVIOR OF THE CONSTITUENTS OF THE MYELIN SHEATH IN SECONDARY DEGENERATIONS. F. GUIDI, Riv. di patol. nerv. **26**: Pts. 1-2, 1921.

The author refers to his previous works on secondary degeneration of the fibers of the sciatic nerve, published in the same journal, in which he demonstrated that the two elements of the sheath (granules and alveoloreticular stroma) are a biologic reality, not artificial staining products, as they do not appear at the same embryogenetic date, but the granules can be demonstrated only many days after the stroma.

In this new series of researches carried out with the same method (author's modification of Besta's method), which he describes in full, the author endeavors to integrate his previous study in order to answer some important questions left unsolved, namely: What are the reciprocal relations governing the granules and the stroma in secondary degeneration? What are the morphologic characters of the degeneration and of the disintegration of the granules? Since the two constituents of the sheath appear at different embryologic dates, will they disappear also at different dates? How is the degenerated material absorbed?

Here are the conclusions at which the author has arrived in this new experimental study which was carried out on the sciatic nerve of rabbits at different dates.

The granular portion of the myelin disappears earlier than the alveoloreticular stroma; it assumes special modifications (metachromatic staining) in the affinity for the staining substances used.

During the degenerative process, the granules show phases which morphologically remind one of the behavior of the stroma; that is the granules become grouped and fused together, the fibers become irregular in their course, are

broken up and gradually disappear. The granules disappear more rapidly than the fragments of the alveoloreticular stroma. The metachromatic staining takes place chiefly in the first phase. The elimination of degenerated granules seems to take place by means of nucleated elements possessing phagocytic powers in which granules are included, probably in order to be directed toward capillaries.

The author is unable to speak definitely concerning the origin of these nucleated elements. He has undertaken a special research on this subject.

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CHICAGO NEUROLOGICAL SOCIETY

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DR. G. B. HASSIN, M.D., *President, Presiding*

A PSYCHOSIS ASSOCIATED WITH EPIDEMIC ENCEPHALITIS.

DR. D. ROTMAN.

A negress, 28 years old, was admitted to the Chicago State Hospital from the Psychopathic Hospital, April 2, 1921. She had marked tachycardia, was extremely restless and profoundly exhausted. Her temperature was 101 F. on admission and had been about 100 F. ever since. The pupils were fixed and angular. There was some misalinement of the eyeballs, hardly enough to be called strabismus. The knee jerks were overactive; there was no Babinski sign. These findings were still present. During an infection of one arm, with a temperature of 104 F., a marked and regular twitch of the left side of the face and of the arm was noted.

The patient had been admitted to the Cook County Hospital, January 1, with marked pain in the shoulder and neck and tremor of the left arm, the same arm that later showed the motor irritation during the arm infection. A tentative diagnosis of neuritis was first made but the appearance in rapid succession of double ptosis, diplopia, lethargy and other symptoms, led to an early diagnosis of epidemic encephalitis. The temperature ranged from 99 to 102 F., the fluid showed 20 cells, the Wassermann reaction was negative. No colloidal gold test was made. Recently both blood and spinal fluid Wassermann reactions had been negative on two occasions; the colloidal gold curve was 1244321000; there were no cells.

Apparently visual hallucinosis and delusions were present from the beginning. She exhibited great fear, thought she was dying and about to pass into hell where she was to be chased by snakes and various animals. On several occasions she failed to recognize her husband.

After nine weeks in the Cook County Hospital, the patient was discharged as convalescent on March 8, but almost immediately she developed marked mental symptoms resembling the preliminary expansive state of a paretic. She believed that she and her family were to visit the folks in the South. They were to do things in grand style. Everybody was to have a new outfit. Asked where the money was to be obtained for all of this, she stated that God had placed the necessary funds in the bureau drawer. She further stated that she was in direct communication with God, and He had told her that if her orders were not carried out the world would be turned topsy-turvy. Taken to the Psychopathic Hospital, she was very much excited, uncooperative and resistive. Since that time the mental symptoms had been ever-changing.

Since admitted to the state hospital she had been disoriented. Unclearness and clouding had been present at all times but had been so fluctuating in degree as to suggest delirium. For short periods she had known the place to be a hospital. At times she had recognized her physician as such, but most of the time she felt herself to be in her home environment. She called the

attending physician Plummer, had never called any one else by this name and had never used any other name for him. One of the nurses who had been with her she called Alice and by no other name, nor did she call any other nurse by this name. This was done by a patient supposedly in delirium.

When first admitted she spit on every one who came near her. This spitting was forceful and seemed purposeful and she would contrive to make it effective. Her explanation was always the same: "I spit on you to save your life." She attacked those about her very much after the fashion of an epileptic in a furor. Unlike an epileptic, however, she was not amnesic for these outbursts. Recently a carpenter had been on the ward making repairs. She forced herself into the room where he was working and was about to snatch the hammer from his hand to strike him. She had remembered this and had mentioned it from time to time with regret, saying "Alice, why did you not let me kill that carpenter?" Some time ago she had broken away from the nurse, had gone to a window, had broken a pane and picked up a piece of glass ready to strike any one who came near, and it was necessary to rush her with a mattress before the glass could be taken away from her. As a matter of fact, this patient had never been physically subdued. Physical restraint had not been successful. She had chewed away the sheets that tied her to the bed and got loose. She had no explanation to offer for these furious attacks.

At times she had remained quiet and seemed to be in a stupor. While in one of these quieter moments a violinist played the chords of "Old Black Joe" for her. She cried immediately and said it reminded her of her father. She then insisted that the musician was her father and called him by name. She then changed from father to son and insisted that the musician was her son. She called to him during the ten minutes he was in the room and begged him to untie his poor mother. This showed how the patient in her delirium was able to weave in some of the things in her surroundings. It also illustrated the peculiar mechanism whereby in her weaving she stopped suddenly and clung to a certain false idea.

A CASE OF ANXIETY NEUROSIS WITH OBSESSION. DR. I. B. DIAMOND.

A young woman, aged 22, an intelligent stenographer, was in bed when first seen because of attacks of palpitation, dizziness and a "sinking feeling as if about to die." The attacks greatly frightened her. She had been nervous for three years, beginning when her mother was very ill. Following this was great disharmony with her office associates and dissatisfaction with her job, which, however, she would not give up because of the liberal pay and easy hours. Then came discontent because she was not married (as were many of her friends) and hopelessness of a good match because so many of the eligibles were in the army. It was easily ascertained that she was sensitive, romantic and a day dreamer. Later and with greater difficulty it was learned that a popular movie actor had been her beau ideal. His physical traits and love making had touched her deeply. She never missed one of his films and identified herself with the heroine with much erotic pleasure. But when she learned that he was much married and the father of numerous children she was disenchanted and gave up his pictures.

Recently she had become engaged and avowed supreme satisfaction with her fiancé. Especially she liked his steady and sedate ways and lack of familiarity. Consequently she was much disturbed by a dream of him; the first dream related to Dr. Diamond. He entered her room disguised as a

villain or thief and his sinister expression so frightened her that she awoke. Then it was learned that a previous (rejected) admirer as a parting shot had said "beware of a Spaniard" and that her fiancé had spent a large part of his life in Spanish countries. Ideas connected with the dream were indicated by the words villain, treachery, Spaniard, seduction, sexual assault. By degrees she admitted that her lover was too close with his money, and that his personal appearance did not please her.

Then she related the following dream: She was out walking with a girl who reminded her of a girl of the neighborhood who had a bad reputation and with whom she had never associated. They bought apples and ate them. Then she went home, was to be dressed in white and was about to die. "Whatever I was going into was regular death to me, against my will." Apparently she was much agitated by this dream and Dr. Diamond's interpretation was that "she is going into or going to do something bad which she fears; namely, marriage, which will lead to her death." Consequently the dream increased her nervousness. After reassurance and explanation that the dream was only symbolic she was much better for a short time. Soon she was in bed again, afraid of consumption and insanity. Now with reluctance she confessed to the obsession that her fiancé had negro blood. When with him she was comfortable, when absent the imperative idea acutely distressed her. Frink (Morbid Fears and Compulsions) says that for American girls the negro is a symbol of sexuality.

Soon after this the patient related another dream. A Chinese woman with a little boy came to the house and wanted a drink of water. Her mother would not let them in, but the patient said "let me get it for her." Then the woman's husband, a big, fat Chinaman, came in, took the patient in his arms and squeezed her. She threw him to the floor and asked her father to put his foot on his neck, but her father (of whom she was exceedingly fond) was indifferent.

Two further episodes in the history were now brought to light, the first apparently forgotten, the second dissembled. Some years before she and another girl in the office, on leaving the ladies' toilet had been confronted by an exhibitionist entirely exposed. They ran away in terror. She recalled that the expression of her lover in the first dream reminded her of this man's expression. It was not true that her fiancé had been uniformly staid and correct. Soon after their engagement she was greatly shocked suddenly to find his hand in her bosom. She was indignant; on his knees he begged her pardon and never again transgressed. Dr. Diamond comments, "it was evident that . . . she had a great fear of the gross sexual (repression) and probably this was the cause of her neurosis." His summary follows.

"We have here a very sensitive and imaginative girl indulging a great deal in romantic day dreaming who received a shock by an exhibitionist. This brings her in conflict with her dream life which she represses with the resulting neurotic symptoms. Her symptoms disappear as she reverts to day dreaming, this time with the help of a motion picture actor. She becomes shocked a second time to find her hero but an ordinary mortal. Her psychic love life is again repressed followed by the return of her symptoms. Unable to make adjustment, she therefore gets into various difficulties in real life. Although 'sick of heroing' as she expressed it, her interest has not been entirely eradicated. The real heroes being sent to war, she therefore regresses to a childish level by her attachment to her father (substitution) which later influenced her love choice. She now receives a third shock by the too ardent wooing of her lover and develops doubts and fears and later obsessions as a

result of her conflicts. Through analysis she obtains a clear insight into her trouble and then makes adjustment to normal which fact she well appreciates. Her comment is worth repeating, 'Doctor, wasn't I the big, silly goose?' It is now almost two years since her illness with no return of her symptoms. She is married and a happy mother."

DISCUSSION

DR. CLARENCE A. NEYMANN looked on the dream essentially as a wish fulfilment. He saw three parts: first, the man who came in with the idea of inflicting some injury; second, the dream of everybody to be feared; third, the dream of the Chinaman, in which the father did not come to her rescue. It seemed to him that the wish fulfilment was not because her lover went too far, but not far enough. He thought Dr. Diamond was dealing with a libido which had not been observed and to which she had not given expression.

DR. RALPH C. HAMILL thought the first dream was of much interest. One frequently meets, in a young girl, the wish for some sexual attention, but she does not want to be responsible for it. In the first dream the man came in as a thief, a housebreaker who was going to do something to her. Dr. Hamill's criticism fell in the same line as Dr. Neymann's; namely, that such a dream indicates a wish for further procedure and yet, perhaps, the more important side was that the patient was not willing to accept responsibility for the wish.

DR. MEYER SOLOMON thought that fundamentally the patient had a neuropathic constitution, and that if one went over her life history carefully it would be found that some of the characteristics were instability, a tendency to undue suggestibility, a constant struggle to come to conclusions about life, and, finally, with her meager knowledge to accept these conclusions as facts. She was at the age where sex problems were not the big thing, and she looked at them as at other things. She had a tendency to flee from life in general, not only from sex problems. The analysis aided her to handle her sex problems directly and to get hold of life more directly. Dr. Solomon wondered whether her recovery was due to the technic that was followed, or whether the analysis did not open up for her a realization of the few defects she had.

DR. HUGH T. PATRICK believed that in this case, as in others of successful psychanalysis, it was not necessarily because the psychanalytic conclusion was the true answer that the girl recovered, but because something was done which satisfied the patient, gave her a new start and helped her to adjust herself. He had no doubt that Dr. Neymann with his interpretation and Dr. Hamill with his conception of the psychology and Dr. Solomon working on the neuropathic constitution, might have been just as successful with the patient as was Dr. Diamond, and it might have been that some old-fashioned doctor, without asking her anything about her dreams, might have gotten along just as well as they with their psychanalysis. He thought the most pertinent thing in the history of the case was the sentence at the end of the paper. "She is married and a happy mother."

DR. SYDNEY KUH said that there had not been more different opinions than speakers, but that he wished to give his interpretation of the case. He felt there was one element back of the case which Dr. Patrick had not mentioned. Dr. Diamond analyzed the girl and explained that all her troubles were due to the nervous instability, but Dr. Kuh thought probably there was a certain hysterical element. She got the impression that this process would relieve her of all her troubles and therefore responded to the treatment.

Dr. William G. Stearns suggested that had the young woman fallen into the hands of an expert osteopath she might have obtained the same results. He believed that all the patient needed was an explanation and assurance that she would get well; that any explanation that would appeal to the patient's sense of logic would cause the same result.

Dr. Julius Grinker said that in a case similar to Dr. Diamond's the patient told him that she was sure that if she had a child she would die, and therefore she had prevented pregnancy. A blind physician had listened to her heart and told her she should not have more children. Dr. Grinker examined her heart and told her that as he could not only hear but also see, he was in a favorable position to assure her definitely that she had no heart lesion; this was sufficient to cure the woman. The patient was not obliged to visit him for weeks and months. Dr. Grinker believed that whatever results Dr. Diamond obtained in his case were due entirely to suggestion. He believed that one must use psychanalysis when examining these patients, examine the workings of their minds and urge them to tell everything that troubles them, and when the physician can throw light on the obscure corners of their psyche and suggest rational modes of viewing a situation—his patient will be cured.

Dr. I. B. Diamond (closing), in reply to Dr. Hamill said that the patient really had a wish for sex attention but was absorbed with the psychic love-life rather than with the physical. The exhibitionist had disgusted her along the physical side, and she was afraid of the whole situation. In the first dream she could not understand why she was so much afraid. He was not thinking of the sex question particularly; the girl was romantic and he had to remove the fears before he could get down to the main complex.

In reply to Dr. Stearns, he would state that the girl had been treated by a gynecologist with tampons for some time and had been treated by a neurologist who tried suggestion, but the patient did not respond to either method. She was cured by obtaining an insight into the cause of her trouble, enabling her to make the adjustment.

OCCUPATIONAL THERAPY IN THE STATE HOSPITAL. Dr. E. A. Foley.

Dr. Foley showed numerous pictures illustrating the work of the occupational department of the hospital.

DISCUSSION

Dr. H. Douglas Singer, state psychopathologist, said that his experience with occupational therapy went back to 1902, but he thought something new had been developed with the work in Illinois. That concerned a realization of what one was trying to do with a better organization. The majority of physicians in thinking about occupational therapy considered it only as something to divert the patient's mind. That was really the smallest part of the present work. He had been in the habit of subdividing the measures used in occupational therapy into three classes:

First, "diversional," in which the effort was merely to direct the mind and keep the patient occupied. It is in this form that it enters largely into the work of a general hospital.

Second, not now used to the extent that it might be, was the form of occupational therapy which he called "symptomatic." As an illustration, there was the man who smokes. Men use smoking to rest themselves. When it

becomes a habit it does not have much toxic effect, but is a form of monotonous activity. Ladies usually take up some other monotonous activity such as knitting, a form of work that requires little attention. There was no question in his mind that the same effect can be obtained by similar means to produce quiet and rest among excited patients. He had known many patients who had discovered this for themselves. Many excited, restless persons like to have something with which to occupy their hands, and so long as they are allowed to do these things they remain comparatively quiet. Other activities requiring more attention with constant change of direction stimulate and increase the patient's interest. By these methods one would apply quieting or stimulating methods which were effective and should be used more commonly.

Third, activities which he considered even more important were contained in the "educational" group. In thinking of one's physiology one realizes that the whole purpose of the nervous mechanism is to do something. The situation here in regard to the occupational methods was much the same as that in the children's school. The child does not go to school, at least in the primary grades, with the idea of acquiring certain information. It does not matter, for example, whether he learns to read or write, for that will come later if he learns to apply himself steadily. Similarly in occupational therapy, it is unimportant what the patient does. The main purpose is to train him to apply himself to some job steadily in such a way as will enable him afterward to meet situations in life.

Dr. Singer expressed strongly the feeling that most important in occupational work are not the articles which the patient turns out. Nothing does more harm than such a view. The real product is the education acquired by the patient. It matters not what the patient does so long as he is trained along lines of steady application, which is essential to social adjustment. The educational side of occupational work is directed toward developing this faculty. At first the occupation must be selected so as to be interesting and attractive to the individual and from that lead up to things not so attractive but more useful for a vocation.

Thence it is important in the early stage to use for occupation activities which are essentially appealing, to make use of bright colors, music and things pleasing to the senses.

In state hospitals it used to be the practice to grade patients according to their disabilities—the restless ward, untidy ward, and so on. Now they are attempting to group patients according to what they can do rather than what they cannot do. Nevertheless, the disabilities must be considered; for example, some place must be provided for the very restless patients where they can be put under more or less symptomatic treatment. But where education becomes possible, the subdivision can be made according to the kind of treatment needed. Thus, some patients' attention is so low that they must be taught the simplest things. Instead of putting them, as used to be done, where they may be untidy if they wish, they are placed where they will be trained to be tidy. The various grades of occupation used for treatment above this simple habit training are selected for the degree of attention which is required to carry them out. As the capacity for attention develops, the patient should be promoted to occupations of increasing complexity.

Finally comes an effort to train the patient for some vocation, either inside or outside the hospital. If occupational therapy corresponds to primary education this feature can be compared with secondary education. Here the actual produce of the patient's activity becomes increasingly important.

Book Reviews

DIE SYPHILIS DES ZENTRALNERVENSYSTEMS; IHRE URSACHEN UND BEHANDLUNG (SYPHILIS OF THE CENTRAL NERVOUS SYSTEM; ITS PATHOGENESIS AND TREATMENT). By WILHELM GENNERICH. Pp. 265. Berlin: Julius Springer, 1921.

Gennerich has produced a monograph on syphilis of the central nervous system considered from the point of view of the syphilologist who looks on central nervous system involvement as merely one incident in the course of syphilis. His is not the point of view of the neurologist; he is not interested especially in symptomatology or nosology or even to any large extent in pathology. What he has done is to attempt to study the life history (Ursachen) of syphilis as it affects the central nervous system from the primary stage until the end of life of the individual. This study is made on the basis of his own tremendous personal experience and deals especially with the way in which the disease may be modified by treatment. It will be recalled that Gennerich has long been identified with the subject of neurorecidive and has entered into the controversy concerning the production of neurorecidive by arsphenamin. We may therefore expect to find him devoting considerable attention in his book to this subject.

A further interest has been the early involvement of the central nervous system, and Gennerich has been one of those who has contributed in large part to our knowledge concerning the high frequency with which the spirochete makes its entrance into the central nervous system during the early stages of the disorder. The book, then, may be said to have a definite philosophy; namely, that of studying the involvement of the central nervous system in all cases of syphilis in an attempt to understand why some cases have serious involvement while others escape with very little clinical symptomatology, and of observing how the various types of treatment affect its course.

The reviewer believes that the book is the most important contribution of its type that has been made. Probably no one else has had the wealth of material to draw on or has kept as satisfactory control over cases for so long a period of time. What is especially pertinent is that he has made a complete, if not universal, study of the cerebrospinal fluid of his cases of syphilis. Before discussing his theories and his contribution in detail one must draw attention to certain therapeutic points peculiar to Gennerich. It was in the summer of 1916 that he first became thoroughly interested in the intraspinal treatment and from that time on he became its most ardent champion. His intraspinal technic is that of the French, namely, the introduction of neoarsphenamin directly into the cerebrospinal fluid, which is then reinjected into the spinal canal. He has been using a routine type of general treatment consisting of a series of eight arsphenamin injections followed by a series of intramuscular injections of mercury. Thus, his whole general treatment of syphilis, whether the nervous system is involved or not, is practically the same until he adopts intraspinal injections. We, therefore, must keep in mind at all times that his conclusions are based on a rather stringent routine which does not, apparently, vary to any large extent from one case to another. He does not use the intensive general treatment or the long continued less intensive injections of arsphenamin so frequently adopted in the United States, nor

has he, apparently, had any experience with spinal drainage, intraventricular, or cerebrosubdural injections, Swift-Ellis technic or the mercurialized serum of Byrnes.

Gennerich states that syphilis of the central nervous system always begins with an involvement of the meninges. In all cases of early syphilis there is "infection of the cerebrospinal fluid," that is, the spirochete makes its way into this fluid. Only a limited number, however, develop clinical symptoms that are discernible. The resultant effect on the individual depends on two things—the immunity reaction of the host and the effect of specific treatment. This immunity reaction as produced by the individual, is, to his mind, the most important concept of all. On this will depend the latter course of the disease, and treatment must be considered from two standpoints, first, what it does to the organism, and second, what it does to the immunity reaction which would normally occur.

Treatment, irrespective of immunity effects, is going to be valuable for the central nervous system in so far as a drug can make its entrance into it. This, according to Gennerich, will depend on the condition of the meninges. He states that the peculiar anatomy and physiology of the pia is of primary importance and that as, in his opinion, the pial surface is nourished by the blood supply only on one side, the effect of such a drug as arsenic is not so satisfactory here as in tissues that have better nourishment. In order to have arsphenamin reach the nervous system it must be in very high concentration in the blood. Illustrating this, he takes up the matter of jaundice, such as one sees frequently after treatment with arsphenamin. He states that one not infrequehtly finds that the pia and the cerebrospinal fluid become colored, but always at a later period than the tinging of the sclera, and that this coloration of the pia remains for a considerable period after the sclera has again become normal. If it is true that it takes a higher concentration of icteric pigment to enter the cerebrospinal fluid and pia, then a high concentration would be even more necessary for a substance like arsphenamin. The condition of the pia determines the amount of diffusion of the drug to a most important extent. Where the pia has been involved by the disease process and has become thickened and changed, the possibility of the diffusion of the drug is greatly lessened. Thus, the cases in which the pia has undergone chronic changes offer the poorest chances for therapeutic results through intravenous treatment.

Metasyphilis is a term which he thinks should be retained to represent the cases of paresis, tabes and the like, and he would classify metasyphilis as a late form of secondary syphilis stating that all cases of metasyphilis begin in the secondary period of the disease. The determining factor in the development of the more benign central nervous system syphilis or the more malignant metasyphilis is the hydrostatic pressure of the cerebrospinal fluid. Where this becomes increased, leading to the diffusion of the organisms into the deep-lying spaces of the nervous system and associated with a considerable pressure degeneration caused by the high pressure, metasyphilis results. These hypotheses are purely logical deductions, and Gennerich gives no physiologic or pathologic data to maintain them. He merely states that they are so. As working hypotheses they may be useful, as they underly his therapeutic attempts and his therapeutic reasoning. It may be said at once that Gennerich is in no way afflicted with a folie de doute; he apparently enjoys complete and thorough belief in his own dogma. These theoretical deductions are the weak points in the development of his thesis and his book. Fortunately, however,

they are only a small part of what he has produced. His work is very completely illustrated by case histories exemplifying and amplifying his statements, and it is on the presentation and grouping of his vast and well controlled material that one must concern oneself in studying his monograph.

He makes his entrance into the study of the subject by considering the results of the treatment of syphilis by mercury and iodids in the pre-arsphenamin days. His general conclusion is that mercury plays very little part in influencing the life process of syphilis in so far as the involvement of the central nervous system is concerned. He finds that there is no great difference in the central nervous system involvement of the patients who had received thorough treatment with mercury and those who had received none. The most that is accomplished is to lengthen the time before the serious damage becomes apparent. However this idea may strike one, it must be said that Gennerich has reviewed the cases of patients treated by mercurials in a most satisfactory fashion, and the conclusions obtained are based on the results. He finds that the results of spinal fluid examinations in patients who had received the classic course of mercurial treatment are far from being as satisfactory as has been generally believed. This work, it seems to the reviewer, is of the greatest importance and must be accepted or dismissed on the results of further study of similar material. Until we do reach a satisfactory conclusion to this matter we have no good basis for an understanding of mercury as an agent in the therapy of syphilis. It may be pointed out, however, at this time that Gennerich's point of attack is not that of the neurologist. Despite the fact that he gives evidence throughout of knowing the difference between the so-called meningo-neurorecidive without clinical symptoms and the more important manifestations which are shown by clinical symptoms, yet throughout he appears to overstress the spinal fluid findings. Thus, in regard to his conclusion concerning the inefficacy of mercury one assumes that he considers abnormal spinal fluid findings of equal importance with actual clinical manifestations. Throughout he seems to indicate that a continued abnormal spinal fluid is merely an early stage of tabes or paresis, but he does not consider the possibility of a person continuing to the end of his days with a positive spinal fluid without any serious mishaps.

Gennerich divides his cases of early syphilis with meningeal involvement into three groups according to the therapeutic results obtained. The first group, representing the vast majority of cases, is made up of those in which the meningeal involvement clears up rapidly with general treatment. The second is a group of cases in which general treatment fails to affect the meningeal process. The cases of the second group will arise despite good intensive general treatment. This raises the comprehensive question of arsphenamin neurorecidives. Is this meningeal involvement the result of irritation by arsphenamin? Gennerich says decidedly that this is not the case, and his chief proof is that these patients recover when treated by intraspinal therapy. All cases, then, of this second group of Gennerich, although developing in the presence of satisfactory general treatment, nevertheless entirely recover when intraspinal injections are given. The third group of cases is composed of those in which the meningeal involvement arises on the basis of a poorly carried out general treatment. These patients are also generally cured by intraspinal treatment. The available space does not permit a complete review of this matter as taken up by Gennerich, but he illustrates very well these three groups with case histories. One wonders whether more intensive general treatment might not accomplish more than was accomplished by Gennerich, but certainly his division of cases is interesting and valuable.

One of the most interesting of the theoretical points is that of the immunity reaction mentioned in the foregoing. According to the author, when the spirochete enters the body it is met by a certain protective mechanism of the patient. This resistance of the body is shown by the generalized symptoms, such as the skin reactions. When these are florid, he believes that the patient is putting up a strong resistance. When the skin reactions are absent the resistance is much less, hence these patients later develop serious disorders such as tabes, paresis, aneurysm and the like. When treatment is given the chemicals tend to check the activity of the infecting organism, but in so doing they tend to cut down the amount of resistance offered by the host. When the treatment is inadequate, that is, when the disease is not cured by killing the spirochetes completely, one is left with a situation in which the resistance of the host is much diminished, whereas organisms are still present in the body and have a chance to develop later and cause serious difficulties. Mercury does not cause such rapid destruction of the spirochete as does arsphenamin, hence the immunity is not diminished to such an extent. He believes that unless treatment with arsphenamin is given vigorously, the results may be worse than if it were not given at all. His statistics, particularly in regard to central nervous system involvement, are exceedingly fertile in showing the effects of smaller and larger amounts of arsphenamin and mercury in the treatment of early syphilis.

Gennerich divides his book into chapters, taking up the treatment of the various forms of syphilis of the nervous system including basal meningitis, meningitis of the convexity, syphilitic epilepsy, tabes, myelitis, paresis and the like. His general thesis concerning treatment is usually simple, namely, that those patients in whom the meninges are quite intact will react well to general treatment. All other patients, and this includes the major portion of patients with symptoms, require intraspinal therapy. By this means he believes the arsphenamin reaches the point of injury.

In reading his text and his cases one is struck by his great optimism as to the results obtained when this method is utilized, and one does not see that he changes his technic to any extent whatever the type of nervous system syphilis with which he deals. Whatever one's point of view concerning the value of intraspinal therapy in the treatment of syphilis of the central nervous system, one is impressed by the results which he reports. His case histories are startlingly interesting. In case after case he shows the development of symptoms and an abnormal spinal fluid despite repeated courses of general treatment, while the same patients improve clinically and serologically to such a degree that they are quite normal after a small amount of intraspinal therapy. One must accept his findings to a certain extent, no matter what one's preconceived ideas may have been. At any rate, it is necessary to look over one's own cases in comparison with his results. He reports the results of treatment under each type of involvement and localization. He maintains that whereas in many cases in which there is a chronic nervous system involvement, symptomatic improvement may occur by the use of general treatment, still these cases will result in paresis or tabes unless the intraspinal treatment is used, in which circumstance he believes the later course will be quite satisfactory. It is feared that his pessimism about the use of general treatment and his optimism concerning intraspinal treatment are both a little excessive being based on a prejudice rather than on a careful sifting of results

He claims excellent and satisfactory results in the treatment of tabes, including cases of optic atrophy and gastric crises. While he does not maintain that the symptoms can be entirely alleviated, he still states that much

improvement can result and the progress be entirely halted. He is also optimistic as to the results of treatment in general paresis. He says that almost every case can be greatly helped, and long remissions of four, five or more years can be obtained in a number of the cases. That more satisfactory results equivalent to a complete cure cannot be obtained in the majority of the cases of paresis is due rather to the damage produced by the hydrostatic pressure of the cerebrospinal fluid than to the direct action of the spirochete. He believes that the activity of the spirochete can be entirely controlled in these cases by the use of intraspinal therapy, but because we cannot modify the pressure changes that have developed and which will continue to develop the results are ultimately unsatisfactory.

One cannot study this work of Gennerich without finding many points for criticism, but, on the other hand, one cannot help but draw the utmost inspiration from his work. The reviewer believes that this is the most thorough study of syphilis of the central nervous system that has yet been offered. It is an attempt really to understand what happens to the patient and what can be accomplished by treatment. Certainly such a study opens the way for a much better understanding of the effects of syphilis on the nervous system than can be obtained in any other way. Many befogged ideas have been thoroughly dispelled by his study of a large number of cases showing what really happens. It seems to us that this is a most monumental contribution and that every syphilologist and neurologist owes it to himself to give this work the most careful study.

KURSUS DER PSYCHOTHERAPIE UND DES HYPNOTISMUS (COURSE ON PSYCHOTHERAPY AND HYPNOTISM). By GEORGE FLATAU. Third Edition. Pp. 175. Berlin: S. Karger, 1920.

The author presents a systematic and detailed review of the subject of psychotherapy. He begins with a brief historical sketch of the treatment of nervous diseases by procedures in which mental factors are the important element. He then proceeds to a somewhat conventional series of psychologic definitions and discussions, after which he takes up the various methods of psychotherapy, devoting the major part of the book to hypnotic procedure.

Dubois treatment and Weir-Mitchell treatment are discussed in a common sense way. In the discussion of the methods of hypnotism the author brings forward no new point of view. He utilizes hypnotism along with the usual common sense hygienic measures, and, although he talks of the hypnotic treatment of masturbation as being "a necessary and often brilliantly efficient means of reinforcement," he furnishes little evidence to show that it is a method which cannot easily be dispensed with.

In the final chapter of the book the author gives a somewhat formal presentation of Freud's psychanalysis with regard to which he is far from enthusiastic.

The book may be recommended to the general practitioner who wishes to know something about the general methods of psychotherapy that are in vogue, but brings little that is new or suggestive to the worker in this special field.